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SOUTH DAKOTA JOURNAL OF MEDICINE

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Volume XXXIV January 1981 Number 1



Clinicopathological Conference
Six Month Old Child With Obesity And Acne

**Isotope Ventriculogram Findings In Hypertrophic
Cardiomyopathy**

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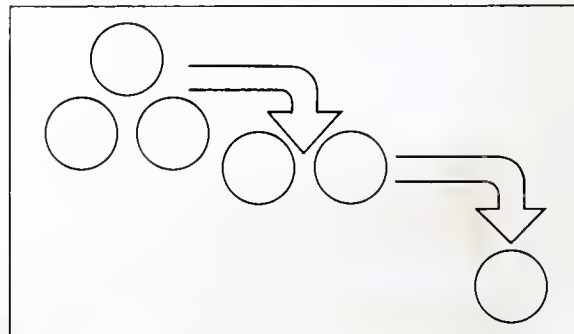
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*Sellers EM: *Drug Metab Rev* 8(1):5-11, 1978



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Six Month Old Child With Obesity And Acne

John Ziemer, M. D.*
Discusser

John F. Barlow, M. D.**
Editor

Case No. S849 516

This 6-month-old female was admitted because of gross obesity. The patient was the product of a full term normal delivery. The mother had gained 40 lbs. in weight during the pregnancy, but there were no known complications. Birth weight was 6 lbs. 14 oz. The marked weight gain had been associated with an increase in appetite requiring 4 oz. of formula every 4 hours for the first two months followed by 7 oz. of formula every 4 hours since then. The patient had been on cereal one month and the child would wake up as many as 5 times a night to eat.

PHYSICAL EXAMINATION: Length 63 cms.; weight 22 lbs. 8 oz.; pulse 120/min. and regular; respirations 28/min. and regular; blood pressure 90 to 100 systolic and 60 to 70 diastolic. The child had a moon facies with extreme plethora. There was a uniform acneform rash over the face, back and trunk. There was no definite weakness in any of the extremities. There were no cataracts or other abnormalities of the head or neck. Lungs were clear to auscultation and percussion. The heart was within normal limits of size with no abnormal sounds or murmurs. The abdomen was obese but no palpable organs or masses were noted. The genitalia were normal prepubertal female. The patient had marked generalized obesity. Neurological examination was intact. (Fig 1)

LABORATORY DATA: Urinalysis yellow, cloudy; specific gravity 1.011, pH 6.0, 2+ protein, negative for glucose, ketone,



Figure 1
Appearance of child (left) compared to normal child (right).

bile, hemoglobin; sediment 0-2 white cells/hpf, 40 to 75 red cells/hpf; repeat urine showed no proteinuria, white cells or red cells on a suprapubic specimen. Hemoglobin 15.8 gms/dl, hematocrit 45 Vol/dl, normal red cell indices, white count 22,000/mm³ with 63% segmented neutrophils, 3% neutrophilic bands, 29% lymphocytes, 5% monocytes. The platelets were normal in number and morphology on smear. A 12-panel showed lactic dehydrogenase (LDH) to be over 600 U/l (normal 0-270 U/l). Alkaline phosphatase, aspartate aminotransferase (SGOT), total bilirubin, total protein, calcium, phosphorus, glucose, blood urea nitrogen, creatinine, uric acid, and cholesterol were within normal limits. pH 7.57. pCO₂ 25 torr, CO₂ content 23 mm/1, sodium 140 mEq/l, potassium 4.0 mEq/l, chloride 108 mEq/l. Urinary free cortisol 205 mcg/24 hrs.

* Resident in Family and Community Medicine, Sioux Falls, SD.

** Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital, Sioux Falls, SD; Professor of Pathology, School of Medicine, University of South Dakota.

(normal 20-120 mcg/24 hrs.) 17 ketosteroids 17.0 mgs/24 hrs (normal 0.3 to 1.0 gms/24 hrs), 17-hydroxycorticosteroids, 12 mgs/24 hrs (normal 1 to 4 mgs/24 hrs). Cortisol AM was 45 ug/dl and PM 40 ug/dl (normal 5 to 25 ug/dl). Adrenal corticotropin was depressed at the same time there was a markedly elevated serum cortisol of 49 ug/dl. Dexamethasone 1 mg. over several days caused no suppression of the serum or urine cortisol, 17-ketosteroids or 17-hydroxycorticosteroids. There was no stimulation of these values by a metyrapone test. Serum insulin, triiodothyronine (T_3) by radioimmunoassay, thyrotrophin and thyroxine (T_4) were within normal limits. Metanephrine and vanilylmandelic acid were within normal limits. Skull films, including polytomography of the sella turcica were negative. A computer tomogram of the abdomen was unremarkable. X-rays of the hands and wrists were within normal limits for age. A chest x-ray was unremarkable. An intravenous pyelogram showed downward rotation of the left kidney suggesting a mass above the kidney to account for the apparent increase in size of the kidney. (Fig. 2) There was medial deviation of both ureters probably related to retroperitoneal fat. A left renal ultrasound showed a mass at the upper pole of the left kidney which did not appear to be cystic. An operation was performed on the 21st hospital day.

DR. ZIEMER: The significant features on the history and physical examination are: (1) large weight gain with generalized obesity; (2) plethora; (3) moon facies; (4) uniform acneform rash (5) high blood pressure for a 6-month-old child.

When this child's weight is compared to the standard female growth curve, it is well above the 95 percentile for the five to six month age group. The description of moon facies, plethora and acneform rash places Cushing's syndrome at the head of the list of the differential diagnosis.

The blood pressure standards for infants are not well defined; however, according to one reference, the 50th percentile for blood pressure for birth to six months would be 80 systolic and 45 diastolic, with an upper limit (95th percentile) at 110 systolic and 60 diastolic. Using these criteria, we can say that this infant does have diastolic hypertension.

The acneform rash described might suggest androgen effect, but there is no description of clitoral hypertrophy which would indicate further consideration of adrenogenital syndrome or other virilizing problems in infancy.

The laboratory data showed a normal urinalysis by suprapubic tap and normal electrolytes. The white count was elevated without any clinical indication of an infectious process. There was also neutrophilia. The only abnormal test on the 12 panel exam was the elevated LDH. LDH levels up to 1500 IU/l can be seen up to six months of age. These tests do not help very much with the differential diagnosis.

Since obesity seems to be the most prominent clinical feature, I would like to begin with this as a differential feature. Obesity can be discussed under three general categories as seen below—genetic, central nervous system, and endocrine.

DIFFERENTIAL DIAGNOSIS OF OBESITY IN INFANCY

- I. Genetic causes
 - A. Laurence-Moon-Biedl syndrome
 - B. Prader-Willi syndrome
 - C. Turner's syndrome
 - D. Frolich's syndrome
 - E. McCune-Albright
- II. Central nervous system causes
 - A. Hypothalamic disease
 - B. Cortical lesions
- III. Endocrine causes
 - A. Insulin producing tumor
 - B. Diabetes mellitus
 - C. Cushing's syndrome
 - D. Thyroid disorder

Laurence-Moon Biedl syndrome is a relatively uncommon disorder. 300 cases have been reported since it was first described in 1865. Obesity is the most common feature with an early onset in infancy. This can be associated with mental deficiency and genital hypoplasia. However, it is impossible to make this diagnosis without other significant features such as polydactyly, retinitis pigmentosa, renal diabetes insipidus, short stature and a family history suggesting an autosomal recessive transmission.

Prader-Willi syndrome was first described in 1956 and 200 cases have been reported. It is associated with short stature and obesity. However, the patients usually have severe hypotonia, small hands and feet, as well as a small penis and nails and mental deficiency. Certainly, this does not fit our case.

Turner's syndrome has been associated with weight gain but this is usually due to marked lymphedema.

Frölich's syndrome is felt to be related to hypothalamic dysfunction which results in obesity, diabetes insipidus, sexual infantilism, short stature and occasionally dysthermia, somnolence, psychiatric disturbance and neurologic signs due to hypothalamic dysfunction often associated with tumor. I feel that we cannot consider this diagnosis.

McCune-Albright syndrome can be included under the endocrine disorders to be considered in this obese child. There are multiple secretions from autonomous endocrine producing glands producing sexual precocity, hyperthyroidism, gigantism or acromegaly as well as Cushing's syndrome. The latter may be the first sign of this entity and is related to bilateral nodular adrenal hyperplasia with a low level of adrenocorticotrophic hormones (ACTH). This syndrome is also associated with polyostotic fibrous dysplasia of multiple bones and café au lait spots on the skin. This entity is quite unlikely here.

The second group of disorders to be discussed in regard to obese children includes central nervous system disorders. Hypothalamic tumors can produce Frölich's syndrome as discussed above. Another syndrome produced is the diencephalic syndrome which can be seen in this age group and is associated with emaciation, hyperkinesis, vomiting, nystagmus, and hypothalamic tumor. Precocious puberty, diabetes insipidus, visual loss and obesity may be accompanying manifestations of the tumor. Cortical lesions have been known to cause obesity, but other neurologic signs should be present.

Among the endocrine disorders which may cause obesity in infancy is insulin-producing tumor which has been ruled out by a normal insulin level. Diabetes mellitus can rarely present as obesity in a child, but with a normal blood sugar and no glycosuria, this diagnosis is excluded. Thyroid disorder is also ruled out by the normal thyroid function tests.

After ruling out all of these entities, one is left with Cushing's syndrome. The abnormalities which we have discussed up to this point and which would be compatible with Cushing's syndrome include obesity, hypertension, plethora, moon facies, acne-form rash, neutrophilia and a suggestion of decreased growth in stature. Children tend to have less hypokalemia and hypochloremic alkalosis than adults. Virilization and hypertension do occur in children with this syndrome. In children, often obesity is less common than weakness, back pain, impaired growth, and hirsutism. In the table below, I am listing some of the common clinical features of Cushing's syndrome.

CLINICAL FEATURES OF CUSHING'S SYNDROME

	AVERAGE %	RANGE %
Obesity	88%	59-100%
Plethora	75%	50-100%
Hypertension	74%	50-90%
Hirsutism	64%	28-93%
Muscle Weakness	61%	18-96%
Menstrual disorder	60%	40-85%
Acne	45%	26-82%
Bruising	42%	23-62%
Mental disorder	42%	31-70%
Back ache (osteoporosis)	40%	22-70%

Cushing's syndrome, by definition, is that clinical picture which is the result of excessive cortisol interaction with the peripheral tissue. The first step in the evaluation of a patient with possible Cushing's syndrome is a screening test to determine the presence of hypercortisolism. This patient's serum cortisol is elevated and there is a loss of the classic diurnal

variation (high values in the morning and lower values in the evening). However, normal patients may have transiently increased cortisol values and patients with Cushing's syndrome may have variable levels. Therefore, there are significant false-positive and negative results when this is the only screening test. Multiple studies have shown elevated values in chronic disease of the heart, liver, brain, kidney and stress states.

A timed urinary collection is the usual answer to smooth out the transient elevations and depressions of the serum cortisol. The free cortisol has emerged as the most reliable of the urinary measurements replacing 24 hour urinary 17-ketosteroids and 17-hydroxy-corticosteroids. Urinary free cortisol is increased due to increased cortisol production, decreased metabolism of cortisol and increased clearance of free cortisol due to the saturation of the plasma protein binding sites. An overall increased clearance of the free cortisol would be expected when there is a general elevation of the cortisol levels above normal.

The third screening test is the dexamethasone suppression test. In children this consists of an 11:00 P.M. dose of dexamethasone (0.6 mgs/L² or 20 micrograms/kilogram) with measurement of a morning serum cortisol. A positive result is a value greater than 7 to 10 ugs/dl.

All of these screening tests are abnormal in this case making the diagnosis almost certain. A patient can often have some positive and some negative test results. These problem situations have been attributed to procedural variables, arbitrary limits for values, hormone metabolism and fluctuations of the cortisol secretion. Although no screening test combination has been tested for diagnostic accuracy, there is a suggested protocol in the **Annals of Internal Medicine** of May, 1979¹. This consists of an elevated plasma cortisol on three occasions, an elevated urinary free cortisol on one of these days, and a positive dexamethasone suppression test.

The next step is to determine the mechanism behind the hypercortisolism. One must rule out exogenous cortisol administration. We have no history for that in this case. There are three endogenous causes of Cushing's syndrome: (1) adrenal; (2) ectopic; and (3) pituitary. Variation in frequency of these in children compared to adults is seen in the table below:

CAUSES OF CUSHING'S SYNDROME

I. Exogenous		
II. Endogenous	Child	Adult
A. Adrenal	65%	8-15%
B. Ectopic	rare	15%
C. Pituitary (Hypothalamic)	30%	70-75%

Adrenal Cushing's syndrome is the most frequent cause in children with approximately 75% of these cases related to adrenocortical carcinoma and about ¼ due to adrenocortical adenoma. There is a female to male ratio of about 2:1. Often there are signs of virilization with clitoral and penile hypertrophy, acne, hirsutism, and osteoporosis in 50% of the cases. The adrenal tumor may be palpable through the abdominal wall. Some adrenocortical tumors are not associated with Cushing's syndrome. Some cases of Cushing's syndrome due to adrenal adenomas have a fluctuating course and, occasionally, an extended remission. A third type of adrenal lesion associated with Cushing's syndrome is called bilateral nodular hyperplasia. It is poorly understood.

Ectopic Cushing's syndrome is very rare in children; and, when it does occur, it is usually related to a malignancy of the thymus or a neuroblastoma or ganglioneuroblastoma. Ectopic Cushing's syndrome is much more common in adults in whom it is often secondary to small cell undifferentiated carcinoma of the lung as well as tumors of the pancreas, thymus, and other organs.

Pituitary Cushing's syndrome is associated with a normal or elevated adrenocorticotrophin ACTH level and bilateral hyperplasia of the adrenals. This represents a significant portion of cases of Cushing's syndrome, both in adults and children. There are still some questions since research into this group has revealed that a pituitary adenoma is present in only 60 to 86% of the cases. An hypothesis for cases without a pituitary adenoma include the hypothalamic production of corticotrophic releasing factor (CRF) at an abnormal rate, which then stimulates increased ACTH.

With improvement in laboratory testing, the radioimmunoassay of plasma ACTH done multiple times in conjunction with a concurrent plasma cortisol has proved to be the most effective laboratory test for distinguishing the various types of Cushing's syndrome. The low ACTH in the presence of a high cortisol is virtually diagnostic of the adrenal form of Cushing's syndrome. This is illustrated by the case under discussion. If the ACTH is within the normal range, the usual source of the elevated cortisol is the pituitary. Very high levels of ACTH are seen in ectopic Cushing's syndrome. Intermediate values of ACTH must be further investigated but can be seen in either pituitary or ectopic causes of Cushing's syndrome. Other tests such as 17-ketosteroids and 17-hydroxycorticosteroids in the urine do not play as important a role as they once did since the improvement of ACTH measurements.

Another test used to differentiate an ectopic from pituitary source of Cushing's syndrome is the high dose dexamethasone test in which there is sup-

pression of ACTH issuing from a pituitary source of Cushing's disease, but there is no such suppression of a similar ectopic source of ACTH. An increase in ACTH after metyrapone is often seen in pituitary Cushing's disease. A third method of distinction can be the measure of jugular vs. peripheral vein ACTH to differentiate a cranial from peripheral source of ACTH. The presence of other peptide byproducts from the synthesis of ACTH may be helpful in the future in indicating ectopic ACTH production.

The third step in the workup of patients with Cushing's syndrome is in the precise localization of the tumor (in this case the adrenal). The intravenous pyelogram only occasionally will disclose a large unilateral tumor. The tumor is, however, seen very nicely in this case (Fig. 2). Arterial or venous radiographic studies can be helpful in localizing adrenal tumors as well. Bilateral cortisol levels in the adrenal veins may be helpful. The computer axial tomographic scan of the abdomen may be helpful but was not in this case. Ultrasonic and nuclear medicine procedures utilizing I-131 Iodocholesterol has been suggested.

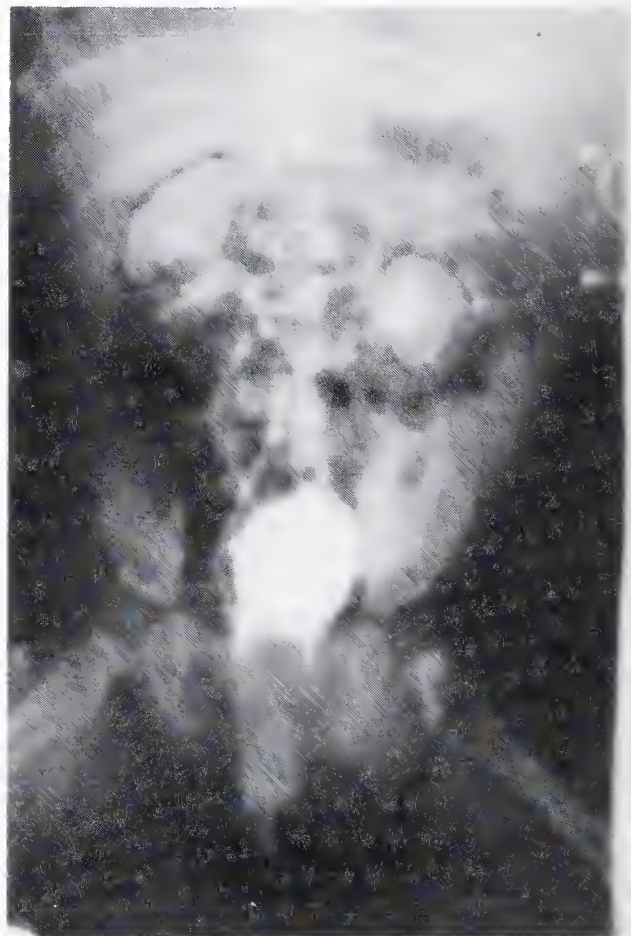


Figure 2
Intravenous pyelogram—the left kidney (kidneys outlined) is depressed by the adrenal tumor.

In regard to treatment—the adrenal form of Cushing's syndrome is best treated by removing the tu-

mor or the bilateral nodular hyperplasia. There are palliative medications which can be used for incurable adrenocortical carcinoma. Long term survival using mitotane (o.p, DDD) or aminoglutethamide have been associated with long term survivals. In this case, after the removal of a single tumor, the patient must have steroid replacement until the other suppressed adrenal recovers. This may take several months. If the patient has not recovered in 12-18 months, it is likely that lifelong replacement with cortisol will be necessary. Since histology is not always accurate in differentiating malignant from benign tumors, a follow-up with urinary cortisol levels on this patient is necessary. I am going to include a final table summarizing some of what I have said:

- I. Screening for hypercortisolism
 - A. Plasma cortisol, 3 days AM, PM
 - B. Urinary free cortisol, elevated at least one of 3 days
 - C. Dexamethasone Suppression Test, 11 P.M. dose, (0.6 mg/m² or 20 ug/kg), A.M. cortisol—less than 7-10 ug/dl indicated a positive test
- II. Determine etiology
 - A. Plasma ACTH
 - B. High dose dexamethasone
 - C. Metyrapone Test
 - D. Jugular vs. peripheral vein ACTH
 - E. Peptide byproducts of ACTH synthesis
- III. Precise localization of lesion
 - A. Pituitary —x-ray sella turcica—tomograms exploratory microsurgery
 - B. Adrenal —intravenous pyelography
—angiography
—CT or ultrasound scan of abdomen
—Nuclear medicine scan with I-131-iodocholesterol
—bilateral venous cortisol
 - C. Ectopic —x-ray—50% of cases show tumor on chest x-ray
—other radiography, edoscopy
—CT scans as necessary

Dr. Ziemer's Diagnoses:
Cushing Syndrome Due To Adrenal Tumor

* DR. NELSON: The patient's adrenal was approached through the anterior abdomen. The left adrenal was enlarged and an approximately 4 cm. tumor was removed which received its entire vascular supply from the aorta and vena cava. The right adrenal was small, measuring 1.5 cm. by 0.5 cm.

DR. BARLOW: Submitted was a 4 cm. mass which on cut section showed a yellow to brown variegated surface. Although the tumor appeared to be encapsulated grossly, (Fig. 3), there was capsular invasion. Other signs of malignancy included marked pleomorphism of the cells as well as mitoses (Fig. 4). The diagnosis of adrenocortical carcinoma was confirmed by the Armed Forces Institute of Pathology.



Figure 3
Cross section of tumor.

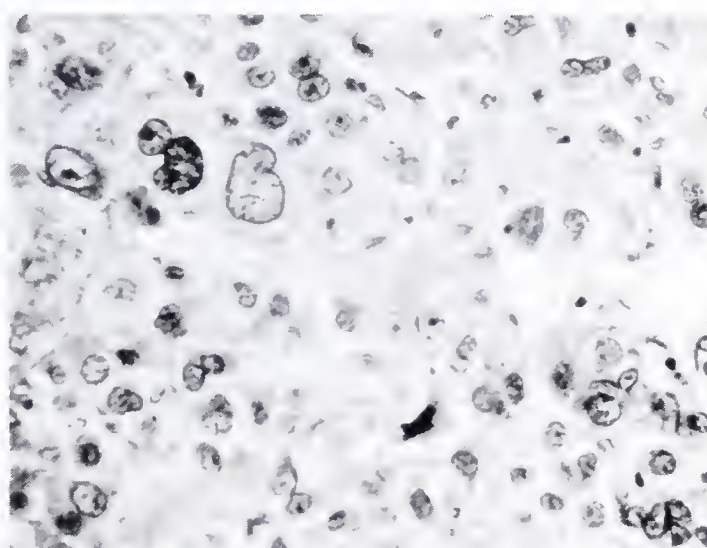


Figure 4
Tumor with marked pleomorphism and mitosis (low center) H & E 450X.

* Surgeon, Sioux Valley Hospital and Central Plains Clinic, Clinical Associate Professor of Surgery, School of Medicine, University of South Dakota.

FINAL ANATOMIC DIAGNOSIS

1. ADRENOCORTICAL CARCINOMA PRODUCING CUSHING'S SYNDROME

*DR. FRED LOVRIEN: I believe Dr. Ziemer's discussion of Cushing's syndrome was very complete. However, there were some specific points which I would like to now address.

One of the questions from the residents was related to why we had done urinary metanephrines. These were done because pheochromocytomas are sometimes seen in association with Cushing's syndrome. It seems logical that excesses of both cortisol, a product of the adrenal cortex, and epinephrine or norepinephrine, products of the adrenal medulla, may be seen in the same patient.

Signs and symptoms of Cushing's syndrome are similar in adults and children, though there are some occasional differences. For example, mental symptoms are quite obvious in adults, but may be very difficult to recognize in infancy. Hirsutism and acne are common to both, and the patient presented today had very striking changes. Both adults and children frequently have truncal obesity. This is often more related to atrophy of proximal muscles rather than to specifically redistribution of adipose tissue. In our patient today, obesity was generalized rather than specifically truncal. This most likely relates to insufficient time for the muscle atrophy to become significant. Striae in Cushing's disease are due to adverse effects of cortisol on collagen tissue. Osteoporosis, on the other hand, is due to the effect of steroids on osteoclasts and because of a negative calcium balance. Neither of these common findings in adults were present in our patient today. The child did have relative hypertension and a uniform acne rash, both quite common in adults. The steroid acne often contrasts strikingly to the more typical acne vulgaris of the teenagers. Steroid acne typically shows lesions all in one phase, where it is much more typical to find lesions in different stages of development in adults with non-steroid induced acne. Lastly, hirsutism is typical of Cushing's syndrome. However, this is generally not the type of hirsutism seen in patients with polycystic ovary syndrome and similar problems. The excess facial hair growth in Cushing's is typically of the lanugo type. This generally shows up as very light, soft hair on the side of the face, rather than the coarse dark hairs on the chin seen in hirsute young women.

In our case today, the laboratory tests for diagnosing Cushing's disease were unequivocal. I just want to add several points about the differential diagnosis of Cushing's syndrome, as Dr. Ziemer has covered this subject quite well. The major categories of hypercortisolism include the ACTH dependent group (primary pituitary disease or ectopic ACTH) and ACTH independent group (primary adrenal neoplasms). Classically, the dexamethasone suppression test has been used to differentiate these forms. In classic Cushing's disease with a pituitary source of ACTH, one will not see suppression on the low dose dexamethasone, but will see suppression on the high dose dexamethasone. Neither ectopic ACTH nor primary adrenal neoplasms will suppress at any level of dexamethasone. However, time has further shown that serum ACTH levels and the response of 17-hydroxysteroids to metyrapone are both better discriminators for elucidating the etiology of Cushing's syndrome than the dexamethasone suppression test. In ectopic ACTH the serum ACTH levels are generally extremely high. On the other hand, primary adrenal neoplasms tend to have extremely suppressed values. Cushing's disease generally has serum ACTH values intermediate between those two extremes. The metyrapone test tends to show an inordinate response in a patient with primary Cushing's disease and generally has minimal or no response in the other forms of hypercortisolism. With the combination of the dexamethasone suppression test, metyrapone test, and ACTH levels, one can generally make a reasonable assessment as to whether the hypercortisolism is from a pituitary etiology, primary adrenal neoplasm, or due to ectopic ACTH.

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A CENTENNIAL NOTE—

The first paid staff employed by the State Association was hired in 1946. John C. Foster was hired as executive secretary. Since that time the Association has had only two other executive secretaries, Richard C. Erickson and Robert D. Johnson. The staff has expanded to a total of seven.

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The specialty organizations recommending the inclusion of these additional procedures include the American College of Physicians, the American Academy of Family Physicians, the College of American Pathologists, the American Academy of Dermatology, the American Academy of Neurology, the American Academy of Pediatrics, and the American Psychiatric Association.

The following is a list of the additional procedures identified:

- Skin test, cat scratch fever
- Skin test, lymphopathis venereum (Frei test)
- Skin test, actinomycosis
- Skin test, brucellosis
- Skin test, leptospirosis
- Skin test, psittacosis
- Skin test, trichinosis
- Autogenous vaccine
- Amylase, blood isozymes, electrophoretic
- Chromium, blood
- Guanase, blood
- Zinc sulphate turbidity, blood
- Cephalin flocculation, thymol turbidity
- Congo red, blood
- Hormones, adrenocorticotropin quantitative animal tests
- Hormones, adrenocorticotropin quantitative bioassay
- Thymol turbidity, blood
- Calcium, feces, 24-hour quantitative
- Starch, feces, screening
- Chymotrypsin, duodenal contents
- Gastric analysis, pepsin
- Gastric analysis, tubeless
- Calcium saturation clotting time
- Capillary fragility test (Rumpel-Leede) (independent procedure)
- Colloidal gold
- Circulation time, one test
- Mucoprotein, blood (seromucoid)
- Calcium clotting time
- Hair analysis test

Bendien's test

Bolen test

Rehfus test

Prolotherapy

Chelation therapy

Cerebellar stimulator pacemakers for cerebral palsy

Hyperbaric oxygen therapy for atherosclerosis

Hyperbaric oxygen therapy for heart attack

Hyperbaric oxygen therapy for cerebral vascular impairment

Hyperbaric oxygen therapy for senility

Hyperbaric oxygen therapy for sickle cell anemia crises

Hyperbaric oxygen therapy for stroke

Orthomolecular medication and megavitamin therapy for use in relation to learning disabilities, mental illness (particularly, Schizophrenia and certain aberrant emotional conditions), hypoglycemia and other non-causally related types of conditions

Intragastric hypothermia using gastric freezing

We do not recommend that physicians categorically discontinue these procedures. Almost every procedure can be medically justified in a specific instance. We do recommend, however, that each physician determine whether the results of any procedure justify the cost.

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The University of South Dakota School of Medicine Alumni Association was officially organized in 1980. It is a self-standing, incorporated organization. It is responsible for alumni programming and raising of funds for general support of the School of Medicine. The Association works closely with the South Dakota Medical School Endowment Association which provides funds for student loans and scholarships and for research support. Both organizations are administered by separate Boards of Directors with assistance from the School of Medicine.

As of 1977 the University of South Dakota School of Medicine is a four-year degree granting school, and through the Alumni Association the school, and past and present students will be better served.

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Modern Ideas On Old Concepts

Somewhere from the antiquious past there is a recurring idea in the minds of physicians that they are not obligated to discuss, before the fact, the financial arrangements for their medical services. I think perhaps the legitimate reasoning involved in this important omission is that the physician has a basic conflict wanting to think of himself as a doer of beneficial things for his fellow man and not wishing to complicate this comfortable idea with the ever present economic design. Scarcely more than a generation ago, when medical care did not loom so big in dollars and cents, there was less interest on the part of the patients to know where they stood prior to engaging the services of a physician. We are aware, of course, that other portions of our economy are equally or even more so inflated in dollar costs, but on the other hand I am sure there are but few of us who would question the awareness that the patients have regarding the high cost of medical care. Some of the more unrewarding developments in professional relationships with patients have been the result of not communicating the financial facts to the patient before care is rendered when such is possible.

Traditionally the medical profession does not change rapidly and this is undoubtedly for the best. I believe we have arrived at the time now where frank discussions regarding the cost of our services should take place, particularly in the area of elective surgeries, non-emergent surgeries or other prolonged and expensive medical care. I am sure this could be one of the factors which would help improve the always increasing incidence of litigation arising out of medical care delivery.

The patient's estimation of what his medical insurance coverage is and what the actual coverage turns out to be is often contradictory. It is in the mutual interest of the physician and his patient, in the non-emergent situation, to find out exactly for the patient what his insurance company is going to cover for the proposed medical care and if the history of the illness could possibly lead to exclusion of the claim by the insurance company. Not only does this relieve some of the tension regarding the patient's care prior to his admission but also leaves the post-care period free of misunderstandings which can lead to break down in the patient/doctor relationship.

When we talk about the currently popularized concept of fee negotiation, there is probably misunderstanding about the definition of this concept. I think we, as individual physicians, would be less likely to want to negotiate with a third party for pre-arrangement of compensation for patient care than we would to negotiate with each patient, not necessarily in a bargaining fashion but in a straight forward presentation, which leads to an arrangement that is made with financial facts on the table. I do not know how many physicians across this state do make it known to the patient the cost of the proposed care and assist in the arrangements for the proposed care, but I think it is a very good idea. It places us in a far better light than letting "the chips fall where they may". Patients may wish to make pre-arrangements for monthly payment and understand better that the fees charged are not contingent upon results. During these discussions, it is also possible to make the patients aware that what they are buying is your sincere application of knowledge and ability to achieve relief for them from their disease. With open approach to the financial aspect of medical care compensation with a patient, there will be less dictation of these arrangements by third parties. It is still acceptable, when it becomes known to the physician that his patient is unable to afford the proposed care, to arrange for him still to have it. Fee reductions, payment schedules, charity work on a voluntary basis cannot be construed as unethical and will serve the interest of patients in need of your services. There is no way in this world at this time that we can avoid addressing these financial arrangements in a straight-forward manner with our patients.

I am off to San Francisco with the delegation from South Dakota to the American Medical Association meeting. I hope to find out some things about the function, organization and policies of the AMA which will be of benefit to our physicians in South Dakota. I would not exactly deny that I will also enjoy myself in the city of San Francisco with all of its culinary and entertaining attractions. Happy New Year!

Sincerely yours,



Winston B. Odland, M.D., President
South Dakota State Medical Association



SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS
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American Academy of Family Physicians

**REPORT ON SURVEY OF
1980 GRADUATING FAMILY PRACTICE RESIDENTS**

The total number of graduates surveyed was 1913. Of this number, 1707 (89.2%) responded. Of these respondents, 1602 indicated type of practice arrangement and 1337 specified the size of the community which they plan to serve. A summary of the results as of July, 1980, follows.

Caution must be exercised in comparing 1980 data with data from previous years because of changes made to data analysis. The data from previous years is being re-analyzed to conform with these 1980 statistics.

PRACTICE ARRANGEMENTS OF 1980 GRADUATING RESIDENTS

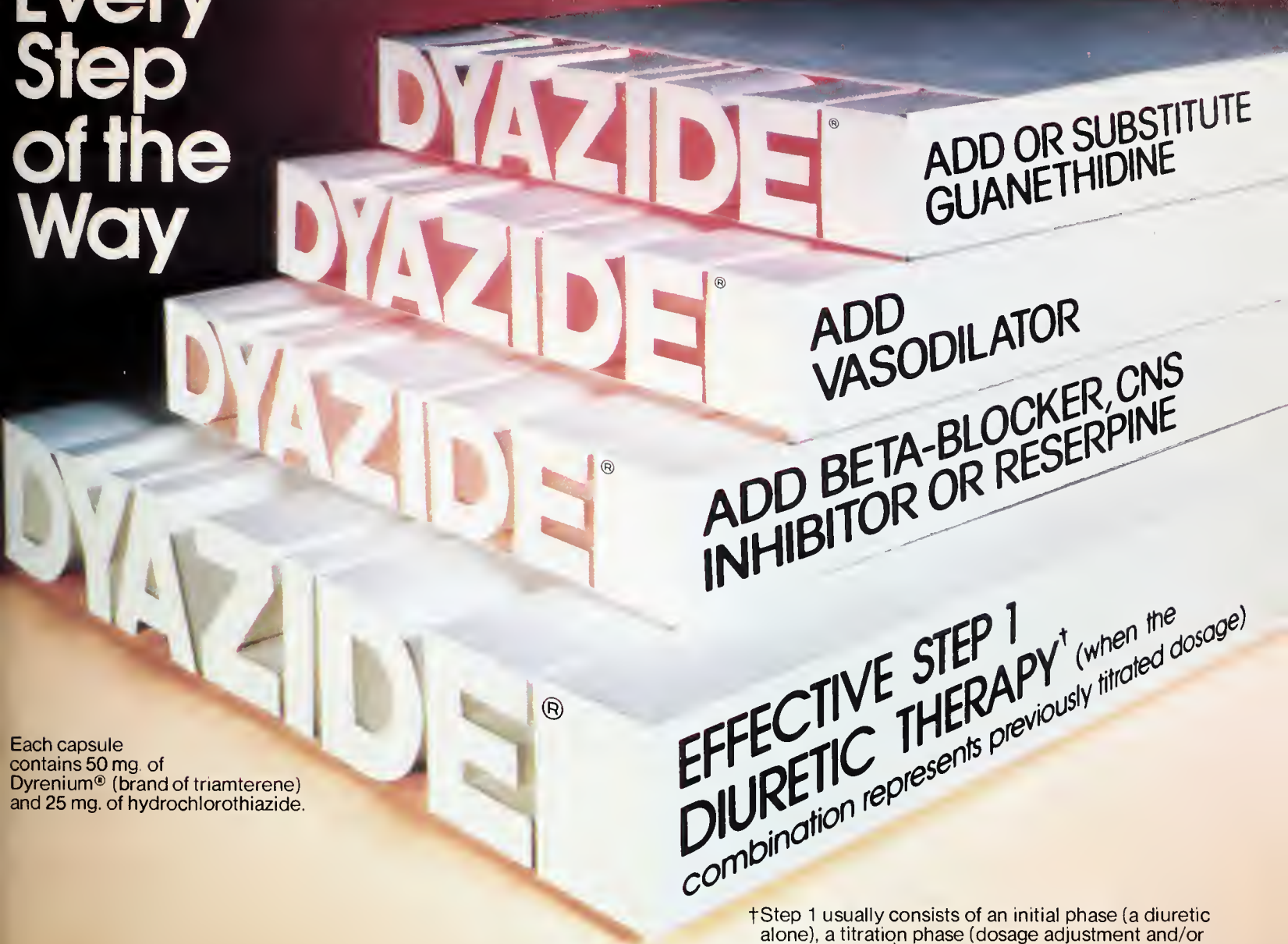
Type of Practice Arrangement	Number of Reporting Grads	Percentage of Total Reporting Grads
Family Practice Group	358	22.3%
Multi-Specialty Group	136	8.5%
Two-Person Family Practice Group (partnership)	283	17.7%
Solo	211	13.2%
Practice (arrangement not specified)	116	7.2%
Military	115	7.2%
Teaching	54	3.4%
USPHS	129	8.1%
Emergency Room	67	4.2%
Hospital Staff	28	1.7%
Research	1	.1%
Administrative	5	.3%
Further Training	33	2.0%
Fellowship	32	2.0%
None of the above	34	2.1%
	1,602	100.0%

DISTRIBUTION OF 1980 GRADUATING RESIDENTS BY COMMUNITY SIZE

Character and Population of Community	Number of Reporting Grads	Percentage of Total Reporting Grads	Cumulative Percentage of Total Reporting Grads
Rural area or town (less than 2500) not within 25 miles of large city	106	8.0%	8.0%
Rural area or town (less than 2500) within 25 miles of large city	37	2.8%	10.8%
Small town (2500-25,000) not within 25 miles of large city	310	23.2%	34.0%
Small town (2500-25,000) within 25 miles of large city	200	15.0%	49.0%
Small City (25,000-100,000)	238	17.8%	66.8%
Suburb of small metropolitan area	50	3.7%	70.5%
Small metropolitan area (100,000-500,000)	134	10.0%	80.5%
Suburb of large metropolitan area	134	10.0%	90.5%
Large metropolitan area (500,000 or more)	78	5.8%	96.3%
Inner city/low income area (500,000 or more)	50	3.7%	100.0%
	1,337	100.0%	

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Each capsule contains 50 mg. of Dyrenium® (brand of triamterene) and 25 mg. of hydrochlorothiazide.

†Step 1 usually consists of an initial phase (a diuretic alone), a titration phase (dosage adjustment and/or addition of a K⁺ supplement or K⁺-sparing agent) and a maintenance phase (a diuretic alone or in combination with a K⁺ supplement or K⁺-sparing agent).

Serum K⁺ and BUN should be checked periodically (see Warnings).

Before prescribing, see complete prescribing information in SK&F Co. literature or PDR. A brief summary follows:

WARNING

This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K⁺ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K⁺ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, throm-

bocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K⁺ frequently; both can cause K⁺ retention and elevated serum K⁺. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Anti-hypertensive effect may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with

possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia, although uncommon, has been reported. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function.

Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components.

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nents alone is rare. (For a complete list of side effects reported with Limbitrol, please consult full disclosure.)

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Please see summary of product information on following page.

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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.
Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.

Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.) Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving).

Usage in Pregnancy: Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline, symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).

Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated.

Sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy.

Limbitrol should not be taken during the nursing period. Not recommended in children under 12. In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.

Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.

The following list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.

Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.

Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.

Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.

Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.

Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.

Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. 1 V administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single h.s. dose may suffice for some patients. Lower dosages are recommended for the elderly.

Limbitrol 10-25: initial dosage of three to four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5: initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.

How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt) — bottles of 100 and 500. Tel-E-Dose® packages of 100, available in trays at 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10. Prescription Paks of 50.

How to initiate and maintain therapy

Select dosage strength appropriate for each patient

- ☐ Limbitrol 5-12.5 is recommended to minimize drowsiness and for elderly patients.
- ☐ Limbitrol 10-25 may be indicated for patients who tolerate medication without undue side effects.

Specify daily dosage based on symptom severity

- ☐ An initial dosage of three tablets is recommended.
- ☐ Dosage may be increased to six tablets or decreased to two tablets daily as necessary.
- ☐ Once a satisfactory response is obtained, patients should be continued on the smallest dose required to maintain the desired effect.

Utilize dosage options to best accommodate individual patient needs

- ☐ T.I.D. or Q.I.D., familiar regimens most suited for patients who tolerate medication without undue drowsiness.
- ☐ Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom — insomnia.
- ☐ Entire dosage h.s. to take maximum advantage of the sedative effect.

Your guide to patient management... when you decide medication is needed

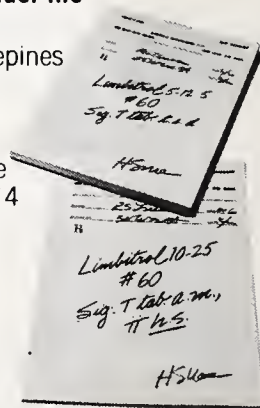
How to make each patient an informed patient

1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.
2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.
3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted.
4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.
5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.
6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information.

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Increase In Physicians Affects Practice Arrangements

Leif C. Beck, LL.B., CPBC*

Vasilios J. Kalogredis, JD, CPBC*

Geoffrey T. Anders, JD CPA*

The Increasing number of new physicians finishing medical school and residency training each year is becoming more and more a factor in health planning. **The Wall Street Journal** recently featured the trend in a front page article exploring the "surplus of MDs."¹ As management consultants and attorneys for medical groups and individual physicians, we are particularly concerned about what this fast-developing trend will mean to our clients.

The subject so far has had little written comment, because it is sneaking up rather quietly on physicians. We consider this article one of the first critical discussions of how the increasing doctor supply actually is affecting medical practice arrangements. These effects vary depending on the age, specialty, and experience of the doctor(s) involved. As the supply/demand situation moves inexorably onward, the effects continue to change.

Hiring a new associate

Medical practices over the past ten years have been generous in hiring new doctors. Other professionals, particularly attorneys and accountants, were hard put to understand their doctor-clients'

hiring at \$40,000 salaries and creating full income parity in just two or three years. Yet the nature of medical practice and the competition for additional well-qualified specialists justified that treatment. We followed the trend by assuming that doctors' starting pay would be at least \$5,000 or more higher each year than the prior year.

Now there is evidence of an opposite trend. Despite continuing inflation, new physicians' starting salaries have not increased particularly over the past couple of years. Offers ranging from \$30,000 in primary care to \$50,000 in various specialties appeared normal in 1978 and again in 1979.

There seem to be reasonable offers for July, 1980 hirings. We wonder if they may continue to be nearly stable into 1981. Salary levels that do not increase by at least 10 percent per year over the several years involved, indeed are lower in terms of real dollars.

The trend which we notice among small medical practices also has been observed at the large, multi-specialty clinic level. In 1979, **Medical Economics** surveyed clinic administrators and found that recruiting well-qualified new physicians was becoming both easier and less costly.² Our experiences with large clinic clients are the same.

Although medical practice decisions must be "economic," just like those of any other business, hiring physicians are not necessarily being selfish in holding down their salary offers. Today, more doctors of the same specialty are "competing" for the

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same patient base. We see a bona fide uncertainty among existing practices as to whether they can expand rapidly enough to afford additional doctor salaries.

In effect, even a busy senior doctor now worries whether he can maintain his own income and pay \$50,000 or more (including increased expenses) to support an associate. As inflation ravages his household and his children's tuitions, the senior doctor understandably resists offering more starting salary than absolutely necessary.

On the other side, the young doctors are finding fewer opportunities to start practices "from scratch" and become highly paid in just a few years. More of them are seeking to join good existing practices at the same time as the seniors are pulling back. A classic example of the law of supply and demand is at work, and it will undoubtedly continue to squeeze young doctors.

One of our employment features may become more useful in view of these circumstances. A senior doctor (or a group of them) unsure whether he can afford a new associate might lower the basic starting salary, thereby reducing the financial risk. In turn, he may promise additional pay once he breaks even financially on the new doctor's involvement—perhaps by paying "incentive compensation" geared to practice gross or net income over the agreed break-even figure. In this manner the senior(s) will have some insulation against risk, while the new doctor will be assured of a better income if he can carry his cost.

The increasing doctor supply also makes "restrictive covenants" much more important to the hiring doctors or groups. Physicians have disdained them in the past as being unnecessary since there were more than enough patients to serve with or without a new doctor's competition.

Now there is concern that an associate who leaves after one year could siphon away some of the practice and that the seniors could not recoup the loss. The senior doctors may be leery of staking a potential competitor as he develops his patients and referrals at his employer's expense. Requiring a promise not to enter competitive practice at the end of the employment, which is enforceable if reasonably drafted, is becoming an important by-product of the changing economics.

Purchase, sale of practice.

The supply-demand factors clearly make successful medical practices increasingly valuable. The physician-owner of a practice can earn considerable income, while others cannot start comparably successful work.

In turn, young doctors may prefer to purchase

existing practices. Seniors then realize that they can sell their practices just like any other items of property. To the extent the purchase price exceeds the value of the physical facilities, it is paid for "goodwill."

In the years of physician scarcity, there was little or no goodwill value to most types of medical practices. We had testified in various courts of law, after considerable survey and research, that many specialties had no goodwill—no one would have "purchased" the practices involved. Young doctors just as easily could have started their own successful offices or joined groups eager for top-flight help; in effect, there was no market.

Even when goodwill value was recognized, the figures were moderate. Many consultants used 20 percent of a year's gross income or 33 1/3 percent of a year's net income as starting points for such valuations.

Now such guidelines are meaningless. Doctors planning to retire have in various specialties asked for prices equal to 100 percent of their practices' annual net income. In a few recent cases the figures have been one year's gross income. Accounting practices have "sold" for one year's gross income over the years, leading us to question whether such medical practice offers were suggested realistically or on the strength of accountants' uncritical and traditional experiences.

Young doctors have at least seriously considered accepting these offers, even though the figures have been refused or negotiated downward.

We no longer feel confident that a practice's goodwill value can be determined reliably. The times seem to be changing so fast on this issue that our advice to senior (selling) doctors is guarded: suggest a considerable price, approaching or even exceeding one year's net income depending on all the circumstances, and then be prepared to hold fast or negotiate downward depending on the young (buying) doctors' responses.

Our advice to the young physician is similarly indecisive: consider how much you really want to practice in the subject area, how good (or slim) your chances are of doing so other than by purchase, how likely other doctors might be to bid on the same practice, etc. You may agree to a high goodwill value for the opportunity to take over a mature practice, paying the price over a period of years; or you can reject it and go elsewhere.

The uncertainty over goodwill has led us to recommend a modification designed to help both sides achieve a fair result. The high value suggested by the senior physician may be reasonable when the practice produces an agreed level of income for the "buyer."

When the practice is less productive for the successor doctor then the purchase price may have been too high. The sale price may be payable over a number of years with a proviso that any annual payment will be reduced or deferred if the continuing practice's gross income falls short of agreed levels. While such an arrangement requires safeguards for the selling doctor, it might offer the young doctor enough assurance to go ahead.

We recognize that the concept of "selling" a medical practice is repugnant to the profession's ideals. A physician undertakes to care for his patients as best serves their needs; transferring them to another doctor for a purchase price may contradict that principle. Nevertheless, the economic reality cannot be ignored.

Young doctors need to find ways to enter practice to use their training and skills profitably. Senior doctors have patient relations which they can pass on to well-qualified successors. Practices thus are being bought and sold increasingly. This trend will continue to grow as the doctor surplus develops.

Promotion to equal partnership

Since practices have increasing goodwill values for outright purchase, it stands to reason that the same factors deserve recognition when newly hired associates are taken into partnership or corporate co-shareholder. We consider the underlying economics for corporations the same as for partnerships. For convenience, this article will not differentiate between partnership and professional corporation status.

A senior doctor who doubts whether his practice can afford a first-year associate's salary will be even more concerned whether it can thereafter double in scope to support an equal income-sharing partner. This fear is merely an added manifestation of the basic concern over the growing doctor supply.

Until recently, a senior physician (or group) tended to promote his associate to equal income parity quickly. The standard arrangement had been to provide the new partner with 60 percent of a full income share in his first year (his second year with the practice, following a year's employment), then 80 percent in the second year and full parity thereafter. The senior partner(s) rarely experienced any drop in actual income during these years.

Now, however, we see a moderation in this willingness to promote quickly. More young doctors are being told that they will reach equal income rights over five years instead of three. The seniors are concerned that their practices may take that long to increase their volumes proportionately. As a result, a new member might now receive 60 percent of a full share in his first partnership year, then 70, 80, 90 and finally equal rights in the fifth such year.

Other arrangements are beginning to take a different approach. Senior doctors are sometimes conditioning the new members' shares upon practice growth sufficient to carry the increasing shares. For example, one high-income specialist took his associate into partnership at a 30 percent share of income, but the young doctor's share would not increase to 40 percent or thereafter to 50 percent until and unless certain agreed practice net income figures were reached. Especially since those target figures increase yearly to recognize inflation, we are not sure whether the young partner will ever reach full parity.

Such arrangements are not necessarily unfair to incoming partners. A senior doctor who has built a successful practice and created a certain income flow may well deserve to continue enjoying it—assuming he continues to give it his full time and attention. Conversely, even if a doctor fresh out of training is capable, it is questionable that he has a "right" to a high income if he cannot attract enough patients by his own devices. In effect again, the increasing doctor supply may thus be creating an income sharing differential to recognize goodwill.

Some people propose that the goodwill item be recognized directly. They would require an incoming partner to pay a substantial lump sum purchase price for partnership. Although this approach has some logic (i.e., if a doctor may sell his entire practice for a price, he may sell half of it to his new partner), usually we do not agree.

We consider goodwill an opportunity to earn continued income which in fact should provide income, even though taxable, to the continuing partner(s). This approach is not particularly undesirable to a senior doctor especially if his higher income carries with it tax-free deflection into a retirement plan for his benefit. And it would permit the new doctor's payment of a purchase price to be tax deductible, which would help him avoid an especially onerous burden. The goodwill element should be recognized by both members so they can proceed to work together in mutual respect and professional confidence despite economics.

Pay to departing partners

Group practices that are costly to join should be comparably more valuable when members die, retire, or otherwise withdraw. We are beginning to advise many of our clients to reconsider their partnership termination provisions in light of changing economics.

Most partnership or group corporation arrangements have over the past ten to twenty years been rather stingy in paying departing members. They have recognized that a partner's death or withdrawal may leave the ongoing partners with a crushing pa-

tient load or else with a new doctor rapidly receiving the same equal income share.

As a result, the pay-out typically was limited to his share of the outstanding accounts receivable, often equated to two or three additional months' pay, plus return of "capital interest" in his partnership or the "book value" of his professional corporation stock.

We are observing and recommending increases in many such distributions. As an example, the group which previously provided only three months' extra pay for a retired or deceased member might amend its agreement to provide a six or nine months' extra pay. The increase, no matter how categorized in the partnership agreement or corporate employment contract, is really a distribution for the goodwill value the departing member will leave behind.

Increasing the termination pay-out may be justified on paper, but the on-going partners naturally will question whether they can afford it. In our sense of priorities, the ongoing practice must be protected. Supporting a departed partner must be secondary to that need. A good approach is to spread the distributions over several years; the ongoing group's monthly pay-out would thus be reduced while the remaining (lesser) income might be paid to a young doctor coming in as a replacement.

Above all, the increasing goodwill value does not justify securing the pay-out with life insurance. Insurance fails to provide the required funds except upon a partner's death, and yet the value should exist whether a member dies or quits.

When goodwill value exists, the practice should be able to provide income enough to pay out a departing member. A group that doubts such capacity probably has no goodwill value. Life insurance should not becloud those underlying economic realities, especially since it requires costly premium payments.

Finally, the goodwill concept makes it important to consider what will happen should a partner withdraw and open a practice in the same service area. Although groups usually recognize a member's right to leave and practice competitively, logically there should be a reduction in his termination pay-out.

In effect, a competing ex-partner will probably have taken a portion of his old group's goodwill value with him in the form of patient contacts and referrals, so he should not also be paid for it. In this case we suggest that the separation pay be reduced to its accounts receivable equivalent, perhaps two or three months' pay, or eliminated altogether if a withdrawing member chooses to compete.

Conclusion

Despite some arguments to the contrary, medical

practices are subject to the laws of supply and demand just as other businesses. The dramatic infusion of medical school graduates and foreign doctors over the past ten years, responding to a perceived shortage of physicians, now is beginning to create a doctor surplus.

Changes in inter-doctor economic arrangements are resulting. We expect the economic realities to continue increasing the importance of a practice's goodwill value.

REFERENCES

1. See "Will surplus of MDs be good for patients? Look at San Francisco," *The Wall Street Journal*, March 13, 1980, p. 1.
2. See "Group practice jobs: Suddenly it's a buyer's market," Harry T. Paxton, *Medical Economics*, November 20, 1979, p. 27-34.

A CENTENNIAL NOTE—

The founding members of the Dakota Medical Society established in 1882 included:

S. B. McGlumphy, M.D., Yankton
O. S. Pine, M.D., Milbank
A. Grant, M.D., Bath
H. G. Rose, M.D., Milbank
L. F. Diefendorf, M.D., Aberdeen
D. F. Etter, M.D., Yankton
W. E. Duncan, M.D., Ellendale
J. B. Van Velson, M.D., Yankton
J. G. Conley, M.D., Elk Point
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Other physicians wished to become affiliated but because the railroad in the Jim River Valley was not yet connected between Aberdeen and Yankton, many were unable to attend the organizational meeting.

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Acute abdominal conditions: The administration of Empirin with Codeine or other narcotics may obscure the diagnosis or clinical course in patients with acute abdominal conditions.

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Special risk patients: Empirin with Codeine should be given with caution to certain patients such as the elderly or debilitated, and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture, peptic ulcer, or coagulation disorders.

ADVERSE REACTIONS: The most frequently observed adverse reactions to codeine include light-headedness, dizziness, sedation, nausea and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include euphoria, dysphoria, constipation, and pruritus.

The most frequently observed reactions to aspirin include headache, vertigo, ringing in the ears, mental confusion, drowsiness, sweating, thirst, nausea, and vomiting. Occasional patients experience gastric irritation and bleeding with aspirin. Some patients are unable to take salicylates without developing nausea and vomiting. Hypersensitivity may be manifested by a skin rash or even an anaphylactic reaction. With these exceptions, most of the side effects occur after repeated administration of large doses.

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Isotope Ventriculogram Findings In Hypertrophic Cardiomyopathy

W. A. Boade, M.D.*

ABSTRACT

Three cases of Hypertrophic Cardiomyopathy (IHSS) are described which were confirmed with cardiac catheterization. Each case had isotope ventriculogram studies performed. The abnormalities of the isotope ventriculogram studies are described.

Hypertrophic Cardiomyopathy (HCM) is a condition in which left ventricular hypertrophy is of unknown etiology. Generally the enlargement of the left ventricle is asymmetric with the interventricular septum more thickened in comparison to the remainder of the ventricle (Idiopathic Hypertrophic Subaortic Stenosis of IHSS). The enlargement of the septum may or may not be symptomatic and may cause obstruction of the blood flow from the left ventricle through the left ventricular outflow tract.^{1,2}

METHODS

We have performed 39 isotope ventriculograms and have found three cases of HCM (IHSS) involving the interventricular septum. Two of these cases have been confirmed with cardiac catheteriza-

tion. ^{99m}Tc labeled red cells are used. This technique requires the IV injection of ionic tin (Sn) as in non-labeled stannous pyrophosphate, followed by the intravenous injection of ^{99m}Tc O₄. The tin (Sn) enables the ^{99m}Tc to attach to the rbc, thus keeping the isotope within the vascular compartment. A standard mobile anger gamma camera and a computer for data processing are employed. The abnormalities were best seen on the cine images, (30°, 45° and LAO) after accumulation of 3,000,000 counts and appropriate computer processing of the data.

Case #1—This 33-year-old white female was referred to the Nuclear Medicine department for evaluation of a heart murmur, which she had known of since age 9. She was on no cardiac medications. She denied shortness of breath, dyspnea, infection, external chest pain, palpitations, orthopnea, paroxysmal nocturnal dyspnea, edema, cough or hemoptysis. She was a gravida IV, para III, AB I with no cardiac difficulties during pregnancy.

Isotope first pass and gated studies utilizing ^{99m}Tc labeled red blood cells were performed. The first pass study showed a low grade left to right shunt. Gated blood pool study showed enlargement of the superior aspect of the interventricular septum. The left ventricular wall motion study showed an abnormality in the region of the septum. The findings suggested HCM (IHSS). (See Fig 1 and 2). Left ventricular ejection was normal (76%). Cardiac catheterization confirmed the above results, with a 20% left to right shunt due to an interatrial septal defect, and enlargement of the upper portion of the interventricular septum.

Case #2—This 21-year-old white man was admitted to the hospital with a history of advanced non-Hodgkin's lymphoma since 1976 with renal failure secondary to obstructive uropathy. There was a history of systolic murmur prior to admission. The patient was receiving adriamycin therapy when referred to Nuclear Medicine for left ventricular ejection fraction determination.

*Specialist in Nuclear Medicine and Pathology, Sioux Valley Hospital, Associate Professor of Laboratory Medicine, School of Medicine, University of South Dakota.



Figure 1

Isotope ventriculogram from Case #1 showing right (rv) and left (lv) ventricular chambers with enlargement of the upper portion of the interventricular septum (ivs).

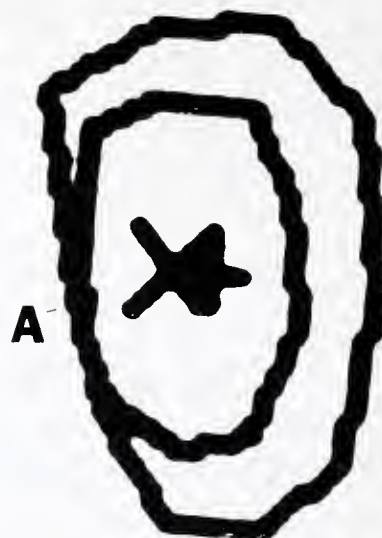


Figure 2

Left ventricular wall motion study from Case #1 showing a septal wall motion abnormality (A).



Figure 3

Isotope ventriculogram from Case #3 showing right (rv) and left (lv) ventricular chambers with enlargement of the upper portion of the interventricular septum (ivs).

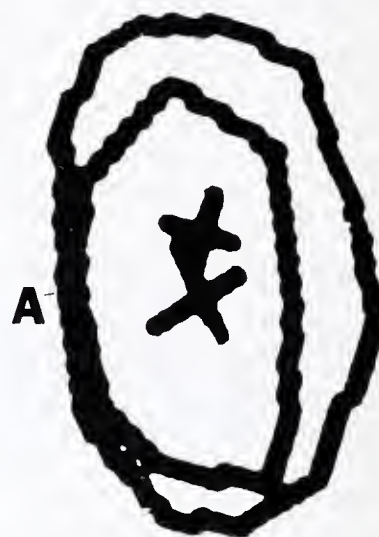


Figure 4

Left ventricular wall motion study from Case #2 showing a septal wall motion abnormality (A).

An Isotope ventriculogram was performed utilizing ^{99m}Tc labeled red blood cells. The left ventricular ejection fraction was well within the normal range (73%). There was enlargement of the interventricular septum superiorly with a wall motion abnormality in the same area. (See Fig. 3 and 4). The patient was not evaluated with a cardiac catheterization.

Case #3—This 24-year-old white female had symptoms of palpitations and shortness of breath and dyspnea on exertion but no definite history of orthopnea, paroxysmal nocturnal dyspnea or pedal edema. There was a family history of heart disease. She had been told she had a heart murmur since birth.

Isotope first pass and gated studies were performed utilizing ^{99m}Tc labeled red blood cells. The first pass study showed no shunt. Gated studies showed prominence of the upper portion of the interventricular septum. The wall motion studies showed

a wall motion abnormality in the region of the mid-to-upper portion of the interventricular septum. Ejection fraction of the left ventricle was normal (82%). (See Fig 5 and 6).

The patient underwent cardiac catheterization and the above findings were confirmed. Echocardiographic studies were also carried out showing widened interventricular septum compatible with IHSS.

DISCUSSION

Three cases of Hypertrophic Cardiomyopathy (HCM) imaged with ^{99m}Tc labeled red blood cell ventriculogram are described. Cine views showed



Figure 5

Isotope ventriculogram from Case #3 showing right (rv) and left (lv) ventricular chambers with enlargement of the upper portion of the interventricular septum (ivs).

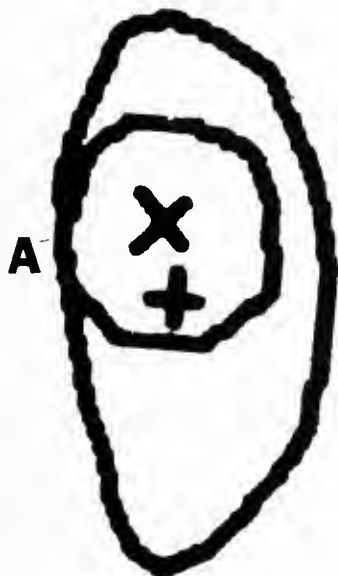


Figure 6

Left ventricular wall motion study showing a septal wall motion abnormality (A).

the abnormality of the upper interventricular septum best at 30°-45° LAO. All cases showed wall motion abnormalities in the region of the interventricular septum along with normal left ventricular ejection fractions. Nuclear ventriculogram can be used to confirm the anatomic abnormalities in HCM along with other techniques. The advantage the isotope ventriculogram shares with the echocardiography is its noninvasive nature.

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2. Epstein, SE, Henry WL, Clark CE, Asymmetric septal hypertrophy, *Ann of Int. Med*, 81; 650, 1974.

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Future Meetings

February

EKG Interpretation and Arrhythmia Management, Hyatt Regency, Phoenix, AZ, Feb. 20-21. 13 hrs. AAFP & AMA Category I credits. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-8646 toll free.

Cardiac Ischemia and Arrhythmias—Current Concepts for Diagnosis and Treatment, Bahia Mar, Ft. Lauderdale, FL, Feb. 27-Mar. 1. 13 hrs. AAFP & AMA Category I credits. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-8646 toll free.

Clinical Management of Coronary Disease and Exercise Testing, Sahara Hotel, Las Vegas, NV, Feb. 27-Mar. 1. 13 hrs. AAFP & AMA Category I credits. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-8646 toll free.

Cardiac Rehabilitation, Hyatt Regency, Atlanta, GA, Feb. 27-28. 13 hrs. AAFP & AMA Category I credits. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-8646 toll free.

March

The Diagnosis and Treatment of Reye's Syndrome, Masur Aud., NIH Clinical Center, Bldg. 10, Bethesda, MD, March 2-4. Contact: Yvonne P. Lewis, Prospects Asso. 11325 Seven Locks Rd., #221, Potomac, MD 20854. Phone: (301) 983-0535.

Advances in Alcoholism, Registry Hotel, Newport Beach, CA, March 6-7. 17 hrs. CME credits. Fee: \$150. Contact: Raleigh Hills Foundation, 17861 Cartwright Rd., Irvine, CA 92714. Phone: (800) 854-3020 toll free.

Clinical Management of Coronary Disease and Exercise Testing, Le Pavillon, New Orleans, LA, March 6-8. 13 hrs. AAFP & AMA Category I credits. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-8646 toll free.

Nineteenth National Conference on Breast Cancer, Hotel Del Coronado, San Diego, CA, March 9-13. 33 hrs. Category I credits. Fee: \$275. Contact: Am. Coll. of Radiology, Breast Cancer Conf., 6900 Wisconsin Ave., Chevy Chase, MD 20015.

Cardiac Ischemia and Arrhythmias—Current Concepts for Diagnosis and Treatment, Sheraton at Fisherman's Wharf, San Francisco, CA, March 13-14. 13 hrs. AAFP & AMA Category I credits. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-8646 toll free.

Clinical Management of Coronary Disease and Exercise Testing, Hyatt Regency, Chicago, IL, March 20-21. 13 hrs. AAFP & AMA Category I credits. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-8646 toll free.

EKG Interpretation and Arrhythmia Management, Aladdin Hotel, Las Vegas, NV, March 27-29. 13 hrs. AAFP & AMA Category I credits. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-8646 toll free.

April

Ophthalmology Clinical Conference, U. of Iowa, Iowa City, IA, April 1. Contact: Richard M. Caplan, MD, Asso. Dean For CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

Otolaryngology Clinical Conference, U. of Iowa, Iowa City, IA, April 3. Contact: Richard M. Caplan, MD, Asso. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

Coronary Heart Disease—1981, Hyatt Regency Milwaukee, Milwaukee, WI, April 9-11. 16 hrs. AAFP & AMA Category I credits. Fee: \$250. Contact: Mrs. Dorothy Black, Pub. Rel. Dept., St. Luke's Hosp., 2900 W. Oklahoma Ave., Milwaukee, WI 53215. Phone: (414) 647-6388.

National Conference on Human Values and Cancer, Washington Hilton Hotel, Washington, D.C., April 23-25. 15 ½ hrs. AMA Category I credits. Contact: Nicholas Bottiglieri, MD, Am. Cancer Soc., Nat'l. Conf. on Human Values & Cancer, 777 Third Ave., New York, NY 10017.

Renal Biopsy Pathology in Medical Disease, U. of Tex. Health Science Ctr., Dallas, TX, April 30-May 2. 22 hrs. AMA Category I credits. Contact: Edwin H. Eigenbrodt, MD, Dept. of Path., U. of Tex. Hlth. Science Ctr. at Dallas, 5323 Harry Hines Blvd., Dallas, TX 75235. Phone: (214) 688-2133.

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Volume XXXIV February 1981 Number 2



Clinicopathological Conference
Two Cases Of Severe Anemia Ending In Death

**Para-Mortem Osteopathology In The Crow Creek
Massacre Victims**

Home Delivery: How Safe?

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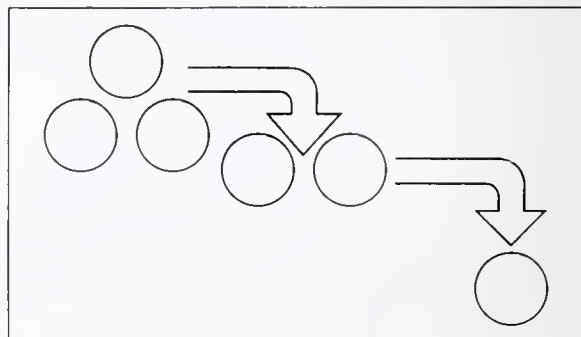
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Para-Mortem Osteopathology In The Crow Creek Massacre Victims*

Larry J. Zimmerman, Ph. D.**

John B. Gregg, M.D.***

Pauline S. Gregg, R.N.****

ABSTRACT

In about 1350 A.D. a group of probable proto-Arikara Indians lived in a village on the Missouri River bluffs in present-day mid-South Dakota. At least 486 of the villagers were massacred. Their remains were buried communally in the fortification ditch which was supposed to have been protecting the north end of the village. This is a brief analysis of the skeletal findings in the Crow Creek massacre victims with special em-

phasis upon para-mortem pathology which indicated vicious warfare and body mutilation by the attackers. Statistical data and illustrative photographs are presented. Evidence of non-lethal scalping, para-mortem scalping, and various forms of skeletal mutilation provide strong evidence that these practices were a part of this culture and definitely antedated European contact. Hypotheses concerning the events surrounding the massacre and possible causes for the catastrophe are advanced.

In about 1350 A.D., a large number of people who lived in the Crow Creek (C.C.) village, located 13 miles north of today's Chamberlain, S.D., were brutally massacred. Their remains were deposited in the west end of the 380 meters long (1250 feet) fortification ditch which protected the north boundary of the village. For a brief interval in 1978-79 the skeletal remnants of the massacre victims were exposed by accident and became available for scientific

scrutiny. Although time constraints and limited budget and facilities precluded all studies which might have been desirable, it was possible to accumulate a very significant amount of data before the skeletons had to be returned to the Crow Creek Sioux Indians.

The archaeologists found a bed of human bones representing men, women and children compressed into an area seven meters square and one meter deep. The temporal bone counts, right- 486, left- 477, provided the most accurate assay for the total number of individuals represented in the common grave (Tables I, II). Because inclement December weather forced the cessation of the archaeological project and the limits of excavation agreed upon

* Paper presented at the annual meeting of the Paleopathology Association, Wednesday, April 16, 1980, in Niagara Falls, N.Y.

** Director, Archaeology Laboratory University of South Dakota.

*** School of Medicine University of South Dakota

**** Sioux Falls, South Dakota.

were reached, it was possible to complete only the west seven meters of the excavation. It is estimated conservatively that there are at least fifty more "burials" remaining in the ground east of the farthest extent of the 1978-9 project. No further excavation is contemplated.

From such an archaeological find it might be hoped that it would be possible to study the entire population of a 14th century village which had existed in mid-America. Unfortunately, due to the almost universally disarticulated and fragmented condition of the skeletons, such information was elusive, other than with certain selected bones.

Almost all of the skulls showed evidence of scalping (Table III), (Figures 1,2). Two even showed reaction on the outer surfaces of the calvaria suggesting the effects of scalping which must have occurred quite some time prior to the ultimately fatal

episode (Figure 3a, 3b). Many skulls had fresh fractures, quite a number of which could have been the death blow (Tables IV, V). The type of fracture in the calvarium in many instances strongly suggested the type of instrument which had been used to inflict the wound (Figures 4,5). The basal portion of many skulls and several upper cervical vertebrae bore markings indicating decapitation (Table VI), (Figure 6). The nose of four skulls bore markings suggesting para-mortem mutilation. The hands were almost totally absent and the feet were missing from the majority of the skeletons, presumably amputated for trophy purposes. (Tables VII, VIII) (Figures 7, 8, 9, 10, 11). Many bones showed evidence of molestation by carnivores (Tables VII, VIII), (Figures 10,11). Although there were more than 700 articulations of two or more bones, it was not possible to identify para-mortem pathology in any total individual.

Table I		
Minimum Element Counts of Major Bones		
Crow Creek Massacre Skeletons		
Bone	Left	Right
Temporal, petrous	477	486
Humerus	200	213
Ulna	113	131
Radius	91	115
Femur	367	367
Tibia	262	269
Fibula	143	156
From: Zimmerman et al, 1980, p. 74		

Table III		
Crow Creek Skeletons		
Cut Marks Upon Frontal Bones Suggesting Scalping		
Bone Bed A.	Definite ---	N = 17----- 94.4%
	Possible ---	N = 1----- 5.6%
Bone Bed B.	Definite ---	N = 254----- 85.5%
	Possible ---	N = 24----- 8.1%
	Uncut-----	N = 19----- 6.4%
Note: Skulls of both sexes and all age groups showed evidence of scalping.		
From: Zimmerman et al, 1980, p. 154.		

Table II			
Smoothed Distribution of Crow Creek Ages at Death			
Age Interval	Males	Females	Total
0- 1	5	4	9
1- 4	16	16	32
5- 9	36	35	71
10-14	20	20	40
15-19	22	7	29
20-24	21	7	28
25-29	16	7	23
30-34	15	7	22
35-39	4	7	11
40-44	3	8	11
45-49	4	13	17
50-54	7	13	20
55-59	6	13	19
Total	175	157	332
From: Zimmerman et al, 1980, p. 10.			

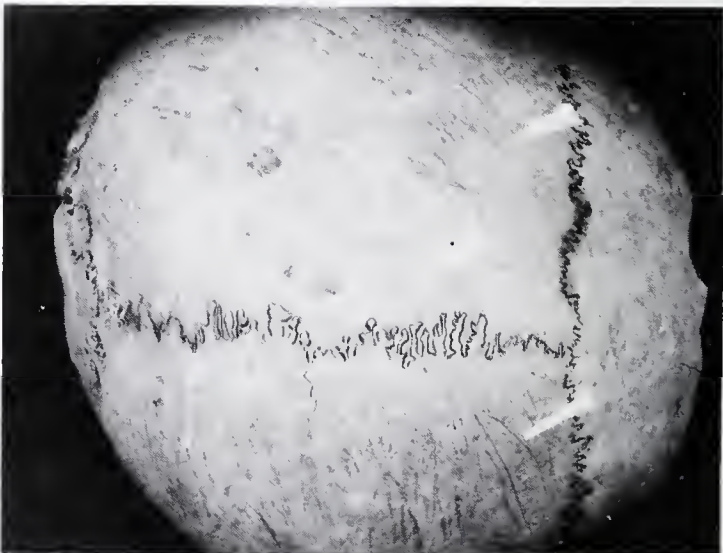


Figure 1
Adult male calvarium, external surface, showing multiple scalping marks, some indicated by arrows.

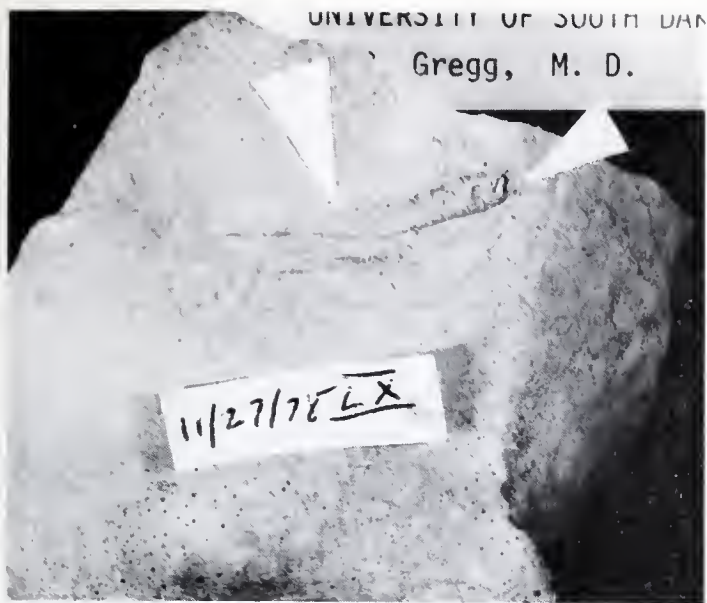


Figure 2
Scalping marks, frontal bone fragment, adult male. Arrows indicate cut marks in the bone and a small flint chip which apparently broke off the scalping knife.

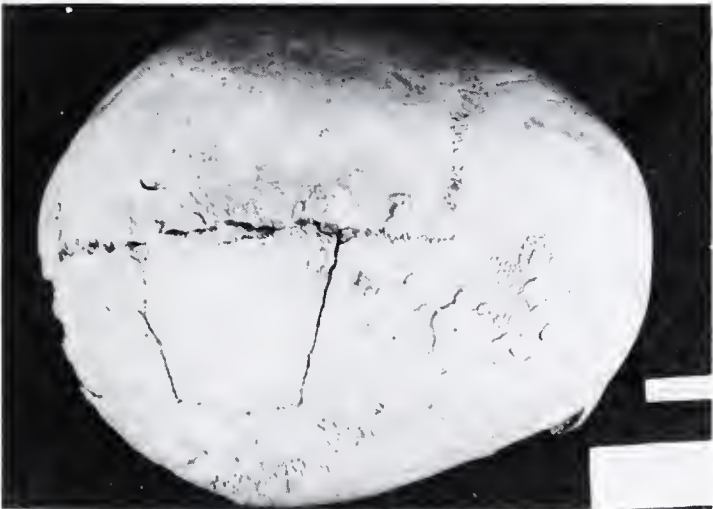


Figure 3 A
External surface of an adult male calvarium. Note the periosteal reaction which is probably the result of non-lethal scalping some time prior to death.

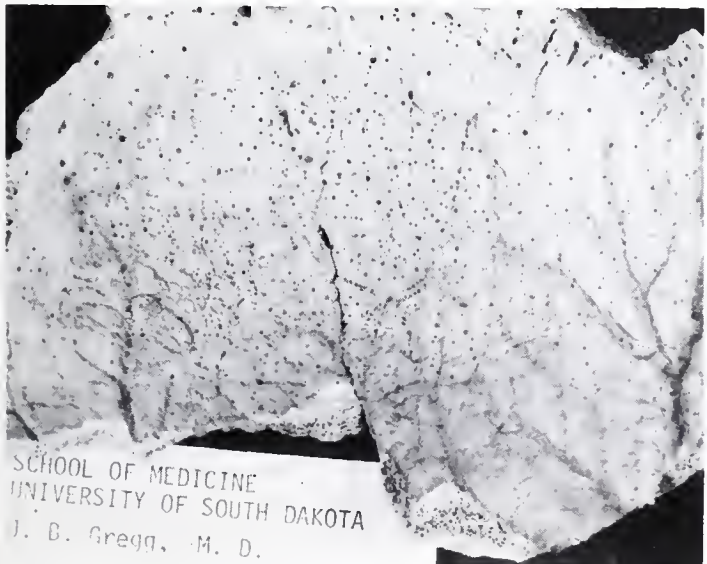


Figure 3 B
Inner surface of calvarium which probably had been scalped some time prior to death. Note the marked dilatation of small blood vessel channels into the bone indicating reparative response to the peri-cranial injury.

Table IV		
Depressed Fractures in 101 Skulls From Crow Creek Massacre Victims		
Number of fractures	Skulls	%
1 -----	28	27.7
2 -----	8	7.9
3 -----	2	1.9
4 -----	2	1.9
5 -----	2	1.9
	42	41.3

From: Zimmerman et al, 1980, p. 156.

Table V		
Location of Depressed Fractures In 68 Skulls of Crow Creek Massacre Victims		
Site	Number	%
Frontal -----	20	29.4
Left -----	18	26.5
Parietal		
Right -----	27	39.7
Occipital -----	3	4.4
	68	100.0

From: Zimmerman et al, 1980, p. 157.

Table VI		
Skulls Showing Evidence of Decapitation Crow Creek Massacre Victims		
Bone Bed A. Occipital—	Definite ---N =	2 --- 66.7%
	None ---N =	1 --- 33.7%
C-1	Definite ---N =	1 ---100.0%
Bone Bed B. Occipital—	Definite ---N =	31 --- 13.6%
	Possible ---N =	6 --- 2.6%
	NoneN =	191 --- 83.8%
C-1	Definite ---N =	56 --- 24.5%
	Possible ---N =	3 --- 1.3%
	None ---N =	170 --- 74.2%
C-2	Definite ---N =	31 --- 16.5%
	Possible ---N =	4 --- 2.1%
	None ---N =	153 --- 81.4%

From: Zimmerman et al, 1980, p. 154.

Table VII				
Alterations In Metatarsals, Bone Bed B, Crow Creek Massacre Site				
Location	Chewing	Snapped Or Splintered	Crushed	Undamaged
Proximal	7 (3.3%)	8 (3.7%)	None	199 (93.0%)
Shaft	4 (1.7%)	30 (12.7%)	None	202 (85.6%)
Distal	21 (11.4%)	48 (26.1%)	11 (0.5%)	114 (62.0%)
Total	32 (5.0%)	86 (13.6%)	11 (0.2%)	515 (81.2%)

From: Zimmerman et al, 1980, p. 163.



Figure 4

External surface of an adult skull showing a fracture defect which was probably made with a blunt, semi-pointed weapon.



Figure 6

Basal view of an adult skull showing pry-marks which were produced during the process of decapitation. Arrows indicate the pry-marks and cuts due to scalping efforts.

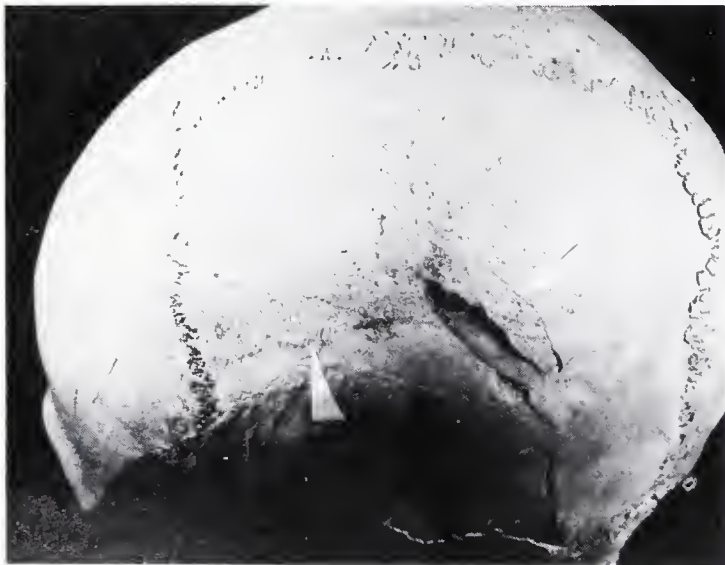


Figure 5

An adult skull with a depressed fracture which was probably made with an elongated blunt instrument, indicated by arrow. The second arrow indicates porosity in the skull which may be secondary to old porotic hyperostosis.

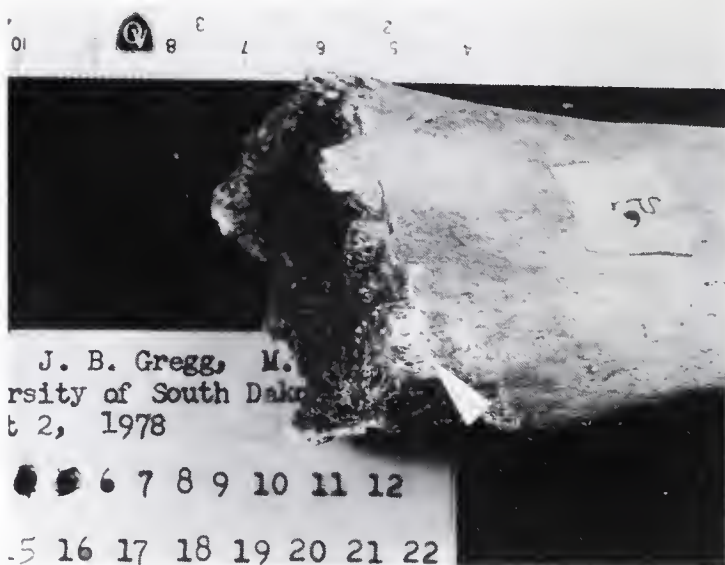


Figure 7

Distal tibia showing evidence of amputation prior to interment. Note soil within the end of the bone. For comparison, a fresh break in the bone produced at the time of disinterment is indicated by the arrow.

Table VIII

Long Bone Modification, Bone Bed B
Crow Creek Massacre Skeletons

Bone	Chewed		Snapped & Splintered		Crushed		Not Modified		Total	
	N=	%	N=	%	N=	%	N=	%	N=	%
Humerous	219	(18.5)	189	(15.9)	8	(0.6)	786	(64.9)	1184	(99.9)
Radius	44	(8.2)	138	(25.6)	4	(0.8)	352	(65.4)	538	(100.0)
Ulna	90	(15.3)	160	(27.3)	9	(1.6)	328	(55.9)	587	(100.1)
Femur	440	(22.4)	219	(11.1)	24	(1.3)	1293	(65.4)	1976	(100.2)
Tibia	160	(11.8)	276	(20.3)	20	(1.5)	908	(66.6)	1364	(100.2)
Fibula	34	(5.0)	115	(17.1)	4	(0.5)	521	(77.3)	674	(99.9)

Instances of questionable identification were omitted.

From: Zimmerman et al, 1980, p. 169.



Figure 8

Facial portion of an adult skull. Arrows indicate scalping marks, old nasal fractures, and fresh cut marks on the left nasal bone.

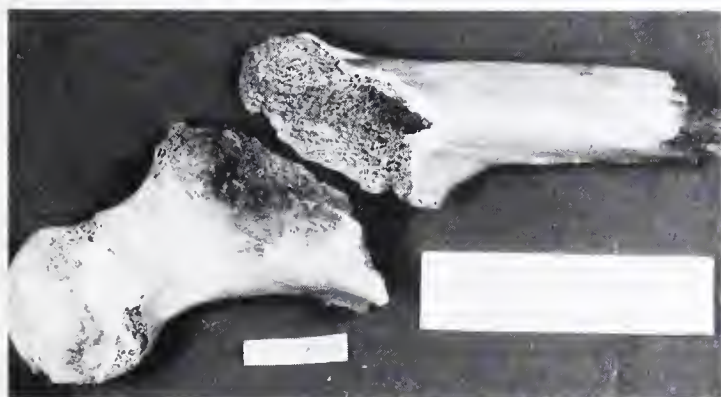


Figure 9

Proximal portion of an adult femur with a post-mortem intertrochanteric break and amputation of the distal portion. Note burn marks in the area of the break and at the distal end. These findings indicate exposure to fire, probably after defleshing.



Figure 10

An adult proximal tibia. Lower arrow indicates rodent gnawings, the upper arrow points to para-mortem cut marks.

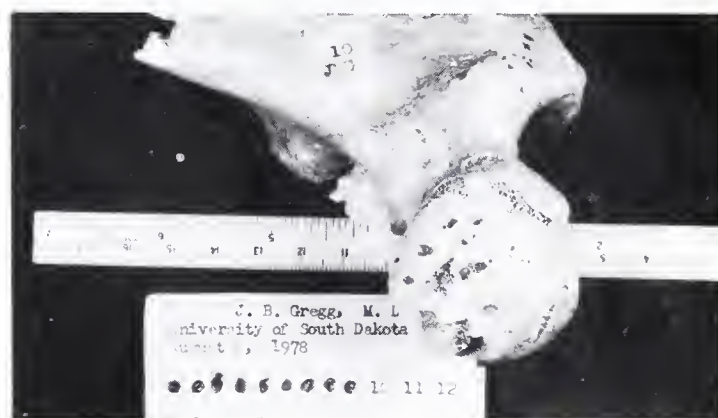


Figure 11

Adult femoral head with tooth marks made by carnivores. Many femoral heads had similar markings, suggesting that this may have been a good point of grasp while dragging the leg.

DISCUSSION

This presentation has been directed toward the discoveries in the C.C. massacre skeletons which might be attributable to events which undoubtedly occurred in the immediate ante-mortem period, at the time of death, in the time period between death and interment, or after the bodies were buried. These findings are related to damage which was done by humans and also by scavenger animals. We feel that information presented here gives damning evidence of vicious warfare and practices which accompanied it in 14th century mid-America. In the future it is planned to present the data relating to ante-mortem osteopathology which was discovered in the C.C. skeletons, indicating the diseases, anomalies and abnormalities which affected them during life.

Because of the fragmented condition of many bones, the disarticulated skeletons, and the absence

of many bones, the **total amount** of para-mortem brutality was not ascertainable. Only specimens with **definite evidence** of para-mortem mutilation are included in this study. In skulls having fractures, because linear fractures could be confused with post-mortem alteration, only fractures which were definitely depressed were included in the tabulation.

The findings relating to scalping indicate that it was definitely a part of this culture and it had no age or sex limitations. Furthermore, scalping per se was not fatal.

Mutilations of victims' bodies, apparently for the purpose of taking trophies, was also a definite part of the cultural characteristics of these people and at this period in time. The findings of the C.C. skeletons **indicate definitely** that scalping and body mutilation antedated White contact.

The C.C. skeletal age/sex distribution (Table II) differs somewhat from that usually found in other Upper Missouri River Basin skeletal populations in that the others usually contain a much higher representation of newborns and very young children (indicating a very high late pregnancy, natal and neo-natal death rate). Although impossible to confirm, the following might be postulated: 1) fewer children at C.C. than in the usual village, 2) abduction of young children by the attackers, 3) escape of some members of the village with many of the children, 4) elimination of the remnants of the young children from the communal "grave" by carnivores or other factors, or 5) burial of the young children at another place in the fortification ditch.

During the archaeological project and the survey for pathology in the C.C. skeletons, **very few** projectile points or evidence of their presence was found. One flint chip, presumed to be the tip of an arrowhead, was found embedded in the lateral surface of an innominate bone, just superior to the acetabulum. Bony reaction around it suggested that it had been **in situ** for some time. Although it might be speculated that arrows were not used in this melee, it is entirely possible that projectiles were extracted from the C.C. victims for future use, or they dropped to the ground as flesh decayed before interment.

The presence of burned bones and findings indicative of molestation by carnivores strongly suggest that at least a portion of the C.C. massacre victims' bodies were exposed for some time prior to "burial" or were exhumed thereafter. Incineration of some bodies or portions thereof is entirely possible. It is highly probable that at least a portion of the bodies of the massacre victims may have been exposed on the prairie for some time prior to burial and that some of the bodies which had been interred

may have been at least partially exhumed by carnivores.

SUMMARY AND CONCLUSIONS

1. At least 486 individuals who lived in a 14th century American Indian village which existed in what is now South Dakota were massacred and their remnants placed into the fortification ditch which surrounded the village. Evidence of the grisly situation became evident by accident in 1978 through soil erosion at the west end of the fortification ditch.

2. There was definite evidence of para-mortem mutilation upon most of the skeletons by humans and scavenging carnivores. Mutilations caused by humans included almost universal scalping and frequent evidence of decapitation, some mutilation of noses, evulsion of teeth, cut marks on many bones, and removal of the majority of the hands and most of the feet. Some bones were burned and carnivore chewing marks were frequent. Missing bones may have been taken as trophies or removed from the village and the burial area by carnivores.

3. It is very possible that at least a portion of the bodies of the massacre victims may have been exposed on the prairie for some time prior to burial and that some of the bodies which had been interred may have been at least partially exhumed by carnivores.

4. The archaeological finding that soil from the river bottom quite some distance from the burial site had been used to cover the decedents indicates purposeful interment of the massacre victims. It is possible that survivors of the catastrophe performed this deed.

5. It is most likely that there were a large number of people in the force which attacked the C.C. village and that they came from a nearby village, probably of the same general cultural group. A night-time attack or some communal illness in the C.C. people which altered their ability to fight or flee may have assured success for the attackers.

6. Reduction in the food supply in the region and subsequent competition for scarce resources are excellent possibilities as the causes for the inter-village warfare.

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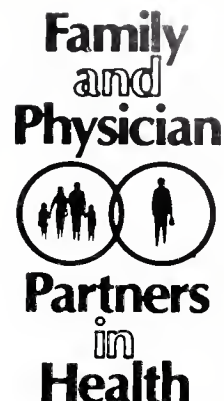
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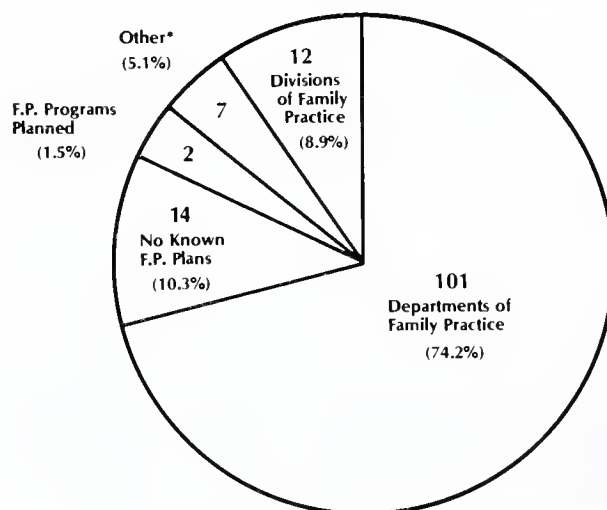
SDAFP Board of Directors, has sanctioned the "SDAFP Memorial Lecture", to be given at each of the chapter sponsored Black Hills Seminars.

This lecture is to be given by an active, affiliate or resident affiliate member of SDAFP on a topic of the speaker's preference. Suggested topics are available from the Education Committee, based upon the cyclic core of knowledge for family practice.

Member applications for the privilege of being selected for this lecture must be available to the state office by April 1 of each year for the Summer Seminar and October 15 for the Winter Seminar. The application will be a letter of intent to be selected and an outline, with references, of the proposed lecture, including the title. The speaker selected for each of these lectures will be handled by the SDAFP Education Committee through a review process.

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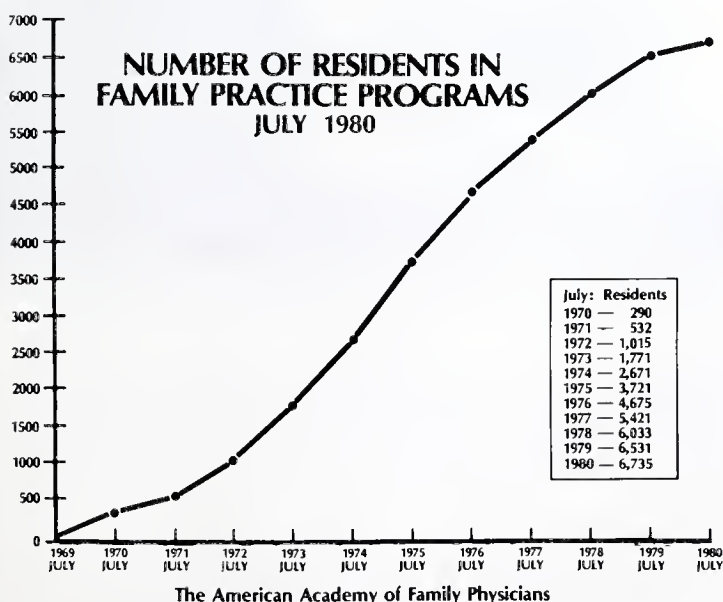
President-Elect Ray Nemer of Gregory received one of six USDSM Special Faculty Recognition Awards at the recent Recognition Days Dinner in Sioux Falls sponsored by the medical school. Congratulations.

Secretary-Treasurer Remains Chairman

L. H. Amundson of Sioux Falls, Secretary-Treasurer of SDAFP, has been re-appointed Chairman of the AAFP Committee on Continuing Medical Education, a committee he has served on since its inception in 1977. He also continues his service on the Commission on Education.

New Bylaws

SDAPR members attending the Annual Business Meeting in Rapid City, August 14, 1981, will be asked to vote on a revised set of Bylaws. This will conform to changes made by AAFP, deleting the Constitution and incorporating it into uniform Bylaws suggested for constituent (state) chapters. A copy of the proposed Bylaws is on file at the state office for members, upon request.



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Home Delivery: How Safe?

Richard R. Thornton, M.D.*

ABSTRACT

The home-birth alternative can be assessed in several ways. First, by reviewing the experience of an earlier era when childbirth was largely conducted in the home, statistics reflect a much greater maternal and perinatal mortality. Secondly, by reviewing preliminary data from current experience with home deliveries, it is clear from this information that perinatal mortality is significantly increased by a factor of two to five times that currently experienced in hospital births. It is also clear that 20% of patients will require hospitalization, either through pre-selection or because of complications that require transfer. Finally, the experiences of obstetricians, as they encounter complications, need to be given some weight in the assessment of the safety of home delivery versus hospital delivery. The data would indicate that complications are difficult to anticipate, but inevitable for a substantial number of parturients.

The writer urges all who hold themselves responsible for pregnant women and their infants to resist the home-birth movement.

Preliminary reports from 47 states indicate that perinatal mortality is two to five times that of hospital births.³ No maternal mortality has been reported to date, but this data is only beginning to be accumulated. Only a few studies are available in this country to assess outcome of elective home births. It is reasonable to assume that the data reported to date should represent the best possible outcome of

The home-birth alternative currently involves 1.09% of all births in the United States. These include 1) accidental deliveries, usually resulting from precipitous labor; 2) certain religious enclaves; 3) poor women, usually from the rural South or migrant workers in the Southeast; and 4) the deliberately selected home delivery.

This paper will deal with the last-mentioned category, namely, the deliberately selected home delivery. The medical profession is currently being asked by a vocal minority to accept this alternative to hospital delivery. The basis for this effort has been primarily attendance or involvement in parturition by family members. A secondary consideration has been cost advantages.

In the last twenty-five years, the safety of parturition has increased remarkably. In 1940, one half of deliveries took place in the home. Maternal mortality was forty times, and perinatal mortality was two or three times the present incidence.¹ Half of all gynecologic surgery was related to childbirth injuries rather than less than 10% noted today.² It is clear from these facts that improvement in maternal mortality and perinatal mortality paralleled the movement to in-hospital delivery.

the home-delivery alternative. One such study, that by Mehl, et al.⁴ reports 1,146 cases. In this group of patients, 16% required hospitalization, either because they were screened out, or because they required transfer to a hospital. Therefore, the best that can be expected is 16%. What is the worst? Are we prepared to embark on a program where perhaps one fifth of patients will eventually be hospitalized anyway? Although this study (Mehl) deals with the subject of perinatal mortality, it does not deal with the possible increased morbidity for the mothers or

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their infants. In a rural state, such as South Dakota, the elements of time and distance must be considered in a system which requires hospital backup. Without the sophistication of hospital care, we can anticipate a return to the 1940 experience.

What data is available in South Dakota childbearing population? What is the ability of competent practitioners to anticipate complications? In a study of perinatal care in South Dakota, it was found by Ranney⁵ that of women encountering complications, over half occurred during labor. 3,360 of these mothers encountered complications. This study included some 10,606 mothers who responded to a questionnaire. In another study by Ranney,⁶ in 2,847 patients identified as high-risk, only 31.9% were identified during routine prenatal visit. Some 62.9% encountered the complication which made them high-risk, either in labor, delivery, or the postpartum period. These were complications which could not have been anticipated; therefore, it is impossible to "screen-out" women for them.

To select one of the more obvious complications for which the home environment is inadequate, it is useful to look at postpartum hemorrhage and retained placenta. In the series reported by Mehl,⁴ there was a 5% incidence of this complication. In an article on retained placenta, Ranney⁷ records a 4% incidence of postpartum hemorrhage in a total of 1500 deliveries. It should be clear that it is impossible to anticipate this complication, and the 4-5% incidence probably represents an irreducible minimum of patients who will require transfer to a hospital in any program of home birth. The responsibility for these patients is then shifted to the attending physician at the hospital. Often the patient's condition has already deteriorated to a point where successful intervention is difficult or impossible. Over the last several years, three such patients, who were delivered in small hospital units lacking blood transfusion and anesthesia capability, were transferred to Sacred Heart Hospital for continuing care. These patients all arrived in various stages of hemorrhagic shock, and required four, five, and eight units of blood respectively. One of the patients suffered sufficiently prolonged hypotension that an isolated gonadotropin insufficiency ensued. This has rendered her permanently infertile and deficient in estrogen. Prichard⁸ in the textbook, **Williams Obstetrics** states that although death from postpartum hemorrhage is rare in current obstetric practice in modern hospitals, it is common under less favorable conditions. If experienced physicians in small hospitals encounter such problems, what then can one expect from home delivery under less favorable circumstances. This is but one example of a number of obstetrical conditions which it is impossible to anticipate and avoid. Others have been pointed out by

Moawad.⁹

How well are hospitals doing? A single maternal mortality has been recorded at Sacred Heart Hospital, Yankton, South Dakota, over the last ten years. This hospital delivers over 800 infants a year. This is fairly representative of national figures which record one maternal death per 8,000 deliveries.

Although some advocates of home delivery emphasize cost, most couples who have home deliveries are actually of substantial means. Furthermore, the questions of where the cost savings is realized, must be answered from a social viewpoint. There can be no reduction in the sophistication or availability of services. Hospital backup is **still** required. In fact, it is not unreasonable to anticipate **more** catastrophic obstetrical occurrences and, therefore, more astronomical hospital bills.

SUMMARY:

From the standpoint of those who practice obstetrics and utilize the surgical, anesthetic, and transfusion capabilities of a well-equipped hospital, the nebulous advantages of home delivery are bought dearly, risking life and health of mother and infant.

It seems appropriate to close this discussion with a reference to the Statement of Policy by the American College of Obstetricians and Gynecologists:¹⁰

"Labor and delivery, while a physiologic process, clearly presents potential hazards to both mother and fetus before and after birth. These hazards required standards of safety which are provided in the hospital setting and cannot be matched in the home situation.

"We recognize, however, the legitimacy of concern of many that the events surrounding birth be an emotionally satisfying experience for the family. The College supports those actions that improve the experience of the family while continuing to provide the mother and her infant with accepted standards of safety available only in the hospital."

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Usage in Pregnancy: Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage, withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline, symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).

Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated.

Sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy.

Limbitrol should not be taken during the nursing period. Not recommended in children under 12. In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.

Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.

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Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single h.s. dose may suffice for some patients. Lower dosages are recommended for the elderly.

Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.

How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt) — bottles of 100 and 500, Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10, Prescription Paks of 50.

How to initiate and maintain therapy

Select dosage strength appropriate for each patient

- Limbitrol 5-12.5 is recommended to minimize drowsiness and for elderly patients
- Limbitrol 10-25 may be indicated for patients who tolerate medication without undue side effects

Specify daily dosage based on symptom severity

- An initial dosage of three tablets is recommended
- Dosage may be increased to six tablets or decreased to two tablets daily as necessary
- Once a satisfactory response is obtained, patients should be continued on the smallest dose required to maintain the desired effect

Utilize dosage options to best accommodate individual patient needs

- T.I.D. or Q.I.D., familiar regimens most suited for patients who tolerate medication without undue drowsiness
- Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom — insomnia
- Entire dosage h.s. to take maximum advantage of the sedative effect

Your guide to patient management... when you decide medication is needed

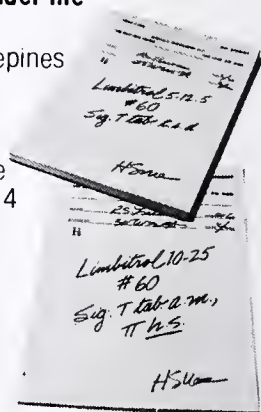
How to make each patient an informed patient

1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.
2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.
3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted.
4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.
5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.
6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information.

Limbitrol should not be used under the following circumstances:

1. Hypersensitivity to benzodiazepines or tricyclic antidepressants.
2. Concomitantly with an MAO inhibitor. To replace an MAO inhibitor with Limbitrol, discontinue MAO inhibitor for a minimum of 14 days before cautiously initiating Limbitrol therapy.
3. During the acute recovery phase following myocardial infarction.



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Two Cases Of Severe Anemia Ending In Death

Carol Z. Dickson, M.D.*
Discusser

John F. Barlow, M.D.**
Editor

Case #845-109

This 51-year-old caucasian male entered Sioux Valley Hospital with chief complaint of shortness of breath and jaundice.

For approximately six months, the patient had noted generalized increasing fatigue and aching in the joints. The patient had had increasing dyspnea on exertion for the past month associated with a knife-like pain between the shoulders which had increased in severity over the past week. It seemed to be relieved by heat and rest. The patient had also noted during the last three or four weeks that his fingers and face would turn blue when he was exposed to cold. This would gradually fade when he was in a warm environment. For the past four days, the patient had noted a dark color to his urine and for one day prior to admission he had noted a yellowish discoloration to his eyes. Six days prior to admission, he had visited a physician for a sore throat and was treated with tetracycline.

The patient was known to have essential hypertension as well as elevated cholesterol and triglyceride levels for some years. He was being treated with propranolol, and a thiazide diuretic.

PHYSICAL EXAMINATION: Alert cooperative man with icteric sclerae and yellow skin, pulse 80/min. and regular; respirations 14/min. and regular, blood pressure 134 systolic and 80 diastolic. Height 5'7", weight 195 pounds. Examination of the head and neck was unremarkable. There was no cervical lymphadenopathy. The lungs were clear to auscultation and percussion. The heart was within normal limits of size. There was a grade II (six grades) systolic soft murmur over the aortic area.

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There were no organs, masses or tenderness in the abdomen. Neurologic examination was within normal limits.

LABORATORY DATA: Urinalysis; orange, turbid, specific gravity 1.020, pH 5.0, negative for protein, glucose, reducing substance, ketone bodies, bile and hemoglobin; sediment, negative. Hemoglobin 6.3 gm/dl; red count, 1,870 million/mm³, hematocrit 18 vol/dl, mean corpuscular hemoglobin 33 micromicrograms, mean corpuscular volume 93 cubic micra, mean corpuscular hemoglobin concentration 35%, white count 11,500/mm³ with 62% segmented neutrophils, 2% neutrophilic bands, 1% eosinophils, 1% basophils, 25% lymphocytes and 9% monocytes. Reticulocyte count uncorrected 4.5%, platelet count 400,000/mm³. Red cells showed gross agglutination on smear and some polychromasia and anisocytosis. When pouring the blood into a test tube, obvious gross agglutination was noted. Rare spherocytes were present on the smear as well as agglutinates of red cells. A cold agglutinin titer was 1/4000 (autoanti I). Free hemoglobin determination in the plasma was normal. A serum haptoglobin was 3 mgs/dl (normal 50-200 mgs/dl). Lactic dehydrogenase 505 IU/L (90-270 IU/L), total bilirubin was 3.4 mgs/dl, 0.4 gm/dl direct and 3.0 mg/dl indirect. Glucose 178 mg/dl, blood urea nitrogen 27 mg/dl; alkaline phosphatase, aspartate transferase (SGOT), calcium, phosphorus, creatinine, uric acid, amylase, sodium, potassium, chloride & cholesterol were within normal limits. A serum electrophoresis & serum immunoelectrophoresis were within normal limits. Test for febrile agglutinins for salmonella, brucella and tularemia were unremarkable. Proteus OX 19 had a titer of 1:40. A serum viscosity was within normal limits. A T4 by RIA was within normal limits. A chest film was unremarkable. Compatible blood was made available after autoabsorption techniques utilizing enzyme treated patient cells.

The patient developed pain in the neck and between the shoulders as well as the classical electrocardiographic and enzyme evidence of a myocardial infarction. He died on the second hospital day.

This 32-year-old caucasian gravid female was admitted to Sioux Valley Hospital for weakness and fatigue. One week prior to admission, she developed what she described as flu with muscle aching and weakness preceded by coryza. She had been seen several days previously by a physician as a prenatal check in the first trimester of pregnancy. A hemoglobin was 10.8 gm/dl. She was placed on iron and multivitamins. She noted increasing fatigue, pounding of the head, ringing in the ears, and shortness of breath on exertion over the next two days, which progressed markedly in the 24 hours prior to admission. There were also episodes of nausea and vomiting without blood in the 24 hours prior to admission.

The patient had been admitted six years previously with a history of lupus-like skin disease characterized by butterfly distribution in the face with involvement also of the earlobes and eyebrows. There was marked sensitivity to sunlight. The rash was erythematous with a bluish tint and numerous small telangiectasia. A biopsy of the ear was diagnostic of chronic discoid lupus erythematosus. On that admission, hemoglobin was 13.6 gm/dl. Erythrocyte sedimentation rate was 26 mm/hr and three L.E. preps were negative. Urinalysis showed a trace of protein, 3-5 leukocytes/hpf, and 1-2 red cells/hpf. A serologic test for syphilis was nonreactive. A total protein was 6.8 gm/dl with 3.7 mg/dl albumin and 3.1 gm/dl globulin.

The patient had a past history of seven spontaneous abortions which had required hospitalization. On all of these admissions, hemoglobin had been over 13 gm/dl and L.E. preparations had been negative on numerous occasions. Serologic flocculation tests for syphilis had been nonreactive.

Prior to the present admission the patient was evaluated at the Mayo Clinic for recurrent pulmonary episodes which were diagnosed as lupus pulmonary reaction. Definite positive L.E. preparations were obtained on follow-up examinations. She had been given steroid therapy, but had not been on steroid therapy for the past several months prior to admission. For a period of several months, she had had muscular stiffness with redness and intermittent heat and swelling in the joints. These symptoms were present all day long.

PHYSICAL EXAMINATION: An obese pale female 5'11" weighing 222 lbs. She was alert and well oriented. Blood pressure was 116 systolic and 72 diastolic. Pulse was 108/min and regular. Respirations were 20/min and temperature 99.4°F. The patient showed marked pallor. There was dusty pigmentation of the face with telangiectasia over the cheeks, but the typical butterfly rash was absent at this time. There was atrophy in the malar region. Lung fields were clear to auscultation and percussion. The areolae were deeply pigmented, characteristic of early pregnancy. She had missed at least two menstrual periods. The uterine size could not be determined because of obesity. There was a presystolic S-4 gallop rhythm present with a rather rough, harsh, midsystolic murmur heard at the apex and left sternal border without specific radiation. No diastolic murmur was heard. Peripheral pulses were symmetrical and of good amplitude. Abdominal examination was unremarkable.

LABORATORY DATA: Urinalysis cloudy, amber, specific gravity—1.015, pH 5.0, negative for protein, glucose, ketone bodies, bile and hemoglobin. Sediment—15-20 WBC/hpf. Hemoglobin 3.5 gm/dl, red count 960,000/mm³, hematocrit 9 vol/dl. Mean corpuscular hemoglobin 37 micromicrograms, mean corpuscular volume 94 cubic micra, mean corpuscular hemoglobin concentration 33%. Total leukocyte count 9,300/mm³ with 71% segmented neutrophils, 6% neutrophilic bands, 1% eosinophils, 21% lymphocytes, 1% metamyelocytes, and 1% nucleated red cell/100 white cells. The red cells showed marked anisocytosis and many spherocytes. The platelets were normal in number and morphology. Reticulocyte count was 25% uncorrected. A direct antiglobulin test was 3+ and an indirect antiglobulin test was 2+. A warm nonspecific antibody was identified. No specific antibodies were noted in the eluate or on dilution. Cold agglutinin titer was 1 to 32. A fluorescent anti-nuclear antibody test (FANA) was markedly positive. 12-panel

chemistry showed normal total protein, calcium, phosphorus, cholesterol, glucose, blood urea nitrogen, uric acid, creatinine serum aspartate aminotransaminase (SGOT) and alkaline phosphatase. A total bilirubin was 1.7 mg/dl. Indirect bilirubin 1.6 mg/dl, direct bilirubin 0.1 mg/dl. A lactic dehydrogenase was 360 units/dl (normal 90-215 units). A chest film was negative. The patient was given 2 units of packed cells which relieved the marked dyspnea. The patient responded to prednisone therapy which was tapered to 30 mg. prior to discharge. She was discharged with the diagnosis of acquired hemolytic anemia, most probably secondary to disseminated lupus erythematosus and an intrauterine pregnancy of 2 months gestation.

One month and a half later the patient was admitted following a spontaneous abortion. She had had a progressive drop of hemoglobin. On this admission, the hemoglobin was 5.6 gm/dl and hematocrit 18 vol/dl. Urinalysis showed no abnormality. A direct antiglobulin test was 2+ and an indirect antiglobulin was 1+. No L.E. cells were observed. She was given 4 units of packed cells which raised the hematocrit to 37 vol/dl. A prominent systolic murmur heard on initial physical examination disappeared after transfusion. A routine serologic test for syphilis was reactive. A fluorescent treponema antibody absorption test (FTA-abs) was nonreactive. Mean corpuscular hemoglobin 36 micromicrograms. Mean corpuscular volume 115 cubic micra. Mean corpuscular hemoglobin concentration 31%. White count 10,100/mm³ with 66% segmented neutrophils, 9% neutrophilic bands, 21% lymphocytes, 1% monocytes, 2% myelocytes, 1% metamyelocytes, 8 nucleated red cells/100 white cells. There was marked anisocytosis and poikilocytosis and marked polychromatophilia. Platelets were normal in number and morphology. An electrocardiogram showed non-specific junctional ST segment changes and the chest film showed a borderline heart size.

Seven months later the patient was admitted because of severe aching of the joints of 48 hours duration. She has been maintained on alternate day prednisone therapy of 80 mgs. She had developed the physical findings of hypercortisolism but not other complications of steroid therapy. The patient had been relatively well until one week prior to admission when she developed upper respiratory infection with cough and some chest discomfort. 24 hours before admission she had felt severe aching in the joints which progressed over the day prior to admission. Physical examination was unchanged over previously. The pulse was 110/min.; blood pressure 136 systolic and 82 diastolic, temperature 100°C. An S-4 gallop rhythm was noted with a sinus tachycardia.

LABORATORY DATA: Urinalysis—yellow, slightly hazy. Specific gravity 1.010, pH 5.0. Trace of protein, negative for glucose, ketone bodies, bile, hemoglobin large amount; sedimentation 0-1 white cells/hpf 0-1 red cells/hpf, no casts. Hemoglobin 8.8 gm/dl, red count 2.65 million/mm³, hematocrit 28 vol/dl, mean corpuscular hemoglobin 33 micromicrograms, mean corpuscular volume 106 cubic micra, mean corpuscular hemoglobin concentration 32%. Total leukocyte count 13,600/mm³ with 70% segmented neutrophils, 4% neutrophilic bands, 23% lymphocytes, 3% monocytes. The red cells were macrocytic with moderate polychromasia. The platelets were normal in number and morphology. A reticulocyte count was 9.9% uncorrected and the erythrocyte sedimentation rate was 115 mm/hr. Direct antiglobulin test was 4+ and indirect antiglobulin test was negative. An L.E. preparation was positive. 12-panel chemistries revealed a lactic dehydrogenase of 340 IU/L units (normal 90-270 IU/L) but all other studies were normal. Because of the continued anemia, the patient underwent a splenectomy without incident on the 5th hospital day. The postoperative course was uncomplicated except for an upper respiratory infection. The hematocrit had risen to 39 vol/dl and reticulocyte count had dropped to 2.3% one week following surgery. The patient was discharged.

Three days later the patient was seen because of cough and shortness of breath for 24 hours. Hemoglobin at this time was 11.5 gm/dl and hematocrit 38 vol/dl. The steroid doses had

been reduced to 30 mgs. of prednisone daily after the operation. Because of a chest x-ray which showed bilateral diffuse infiltrates, the patient was admitted. Significant changes in physical examination included a blood pressure of 94 systolic and 70 diastolic, temperature of 101.6° and pulse 136/min. and regular. The patient showed cushingoid appearance with moon facies. She had a slight buffalo bump and rotund belly with striae. There were crepitant rales in the anterior lung fields which were "dry". There was no friction rub. The heart was not enlarged. There were no murmurs.

LABORATORY DATA: Urinalysis—straw, cloudy. Specific gravity 1.007, pH 6.0 trace of protein, negative for glucose, reducing substances, ketone bodies and bile; hemoglobin small amount; sediment 2-4 white cells/hpf, 3-5 red cells/hpf; hemoglobin 9.8 gm/dl, hematocrit 31 vol/dl. Total leukocyte count 20,300/mm³ with 88% segmented neutrophils, 5% neutrophilic bands, 6% lymphocytes. There was toxic granulation of the segmented neutrophils. pH was 7.47, pCO₂ 27 torr, CO₂ content 19 mm/L, sodium 131 meq/L, potassium 4.6 meq/L, chloride 93 meq/L, pO₂ 61 torr, O₂ saturation 92%. Sputum culture showed normal flora. Total protein, inorganic phosphorus, cholesterol, glucose were normal. Calcium was 4.8 mg/dl, total bilirubin 1.8 gm/dl, indirect bilirubin 1.6 mg/dl, direct bilirubin 0.2 mg/dl. Alkaline phosphatase 198 units (normal 20-90 units). Lactic dehydrogenase (LDH) markedly elevated, aspartate aminotransferase (SGOT) 112 units/dl. A chest film showed diffuse interstitial and alveolar infiltrates over all portions of both lung fields. The patient was placed on oxygen therapy. Follow-up arterial gases showed no change in arterial oxygen levels. The CO₂ content, however, dropped to 13 meq/L. Patient was continued on steroid therapy in the form of 30 mg daily and started on minocycline. Erythrocyte sedimentation rate reached 122 mm/hr but the hemoglobin remained stable at 9.4 gm/dl, and hematocrit 29 vol %. The patient died two days after admission.

DR. CAROL DICKSON: Both of these patients are excellent examples of the two major varieties of autoimmune hemolytic anemia.

The diagnosis of a hemolytic process can be made by: 1) excluding active bleeding; 2) searching for evidence of increased red cell destruction and 3) documenting evidence for compensatory erythropoiesis. Neither of these patients had evidence for active bleeding. Decreased haptoglobin levels, increased lactic dehydrogenase (LDH) as well as increased indirect or unconjugated bilirubin were present. Some patients may develop hemoglobinemia and hemoglobinuria. Both patients had definite evidence of compensatory erythropoiesis as indicated by sustained reticulocytosis. The reticulocytes are young red cells which can be noted on special stains. They are indicated in the peripheral blood smear as polychrome cells. The reticulocyte count should be corrected for anemia. This is done by multiplying the reticulocyte count in percent by the patient's hematocrit divided by the normal hematocrit level. Even when this is done, the reticulocyte count remains elevated indicating an erythroid hyperplasia of the bone marrow in the cases discussed here. Once a diagnosis of hemolysis has been established, the cause for the hemolysis should be determined. A very helpful test in this regard is the direct antiglobulin test. If this test is negative, one

might consider one of the hereditary hemolytic anemias such as hereditary spherocytosis, a hemoglobinopathy, or thalassemia. A large spleen trapping red cells (hypersplenism) or a mechanical nonimmune cause of hemolytic anemia commonly called microangiopathic could be the cause. In the latter case, schistocytes (many broken up red cells) are seen in the peripheral blood. However, we are fortunate in this case that the direct antiglobulin test is positive. As the antiglobulin test indicated an antibody on the red cell, we have indicated the presence of an immune hemolytic process and probably autoimmune hemolytic anemia. Two types of antibodies produce this disease, one which acts at 37°C and is called a warm antibody and another which reacts at 4°C to 20°C and is called a cold autoagglutinin. Either of these two types of antibodies may occur as a primary or idiopathic cause of anemia with no ready explanation or they may be seen secondary to a variety of conditions such as lymphoma or collagen disease. One of our patients today has a warm antibody autoimmune hemolytic anemia secondary to lupus erythematosus. An example of a secondary cold antibody autoimmune hemolytic anemia is seen after mycoplasma pneumoniae.

Besides primary or secondary autoimmune hemolytic anemia due to warm or cold antibodies, a third type is caused by a variety of drugs including the penicillin, the cephalosporins, quinine, and methyldopa. Drugs do not seem to be implicated in this case.

The first patient is a 51-year-old gentleman who presented in the hospital with symptoms associated with acquired cold agglutinin disease resulting in hemolytic anemia leading to severe cardiac decompensation and death. It could be postulated that his anemia began six months prior to admission with symptoms of fatigue. This latter progressed to anginal chest pain and dyspnea on exertion as his cardiovascular system could no longer compensate for the increased stress imposed by the progressive anemia. The arthralgias and acrocyanosis associated with Raynaud's like phenomenon are characteristic of cold agglutinin disease. Typically symptoms improve on warming. This phenomenon seems to be the result of in vivo agglutination produced by the typical IgM molecule attached to the red cell. The attraction of complement to the system may bring about hemolysis. It is, however, the vascular obstruction by the small hemagglutinates forming at low temperature in the distal extremities which produces the symptoms. The agglutinates are reversible on warming.

Jaundice and hemoglobinuria are manifestations of the hemolytic process which is the result of the

lytic complement damage producing red cell fragmentation and phagocytosis. As I have state above, cold agglutinin disease may be either idiopathic or secondary to some disease process. The idiopathic variety is seen in more elderly patients and often takes a more chronic benign course. The secondary variety is often secondary to a lymphorecticular malignancy. Secondary cold agglutinin disease may also follow viral disease such as influenza, infectious mononucleosis or mycoplasma pneumoniae infections. These infections are thought to stimulate the growth of clones of lymphocytes producing specific cold agglutinins. These infectious causes of secondary cold agglutinin disease are often seen in younger patients and are usually self-limited, lasting from weeks up to a few months.

The diagnosis of cold agglutinin disease is rapidly confirmed by laboratory methods. Typically a normochromic, normocytic anemia is present with reticulocytosis. Spherocytosis in varying degrees can be seen in the peripheral smear as well as erythrophagocytosis by monocytes. There is often mild leukocytosis if there is very active hemolysis.

Although cold agglutinins are present in normal individuals, in patients with cold agglutinin disease, the autoagglutinin titers are very high. One can easily note spontaneous agglutination of blood on a slide or a tube which is slightly cold. This is reversed on warming. The diagnosis is confirmed at this point. The agglutinins isolated are often of anti-I specificity. These are IgM immunoglobulins. Interestingly, they are often monoclonal IgM kappa proteins. This could make one think of a malignant disease, but this has not been proved. Frequently the agglutinins are capable of fixing complement and thus intravascular or extravascular hemolysis which is accompanied by indirect bilirubinemia, elevated LDH levels, and depressed haptoglobin levels.

In the benign form of cold agglutinin disease, avoidance of the cold will often relieve the annoying peripheral symptoms. However, acute hemolysis may occur as illustrated in this case. Steroids have generally been found ineffective. Splenectomy is usually of no benefit as the erythrocytes are destroyed in the liver in cold agglutinin disease. There has been some success in treating these patients with cytotoxic agents such as chlorambucil or cyclophosphamide. These drugs have often decreased the level of cold agglutinins.

Blood transfusions may be needed in life threatening cases. It is important to transfuse just enough to keep the patient out of significant cardiac embarrassment and to allow other forms of treatment to bring the patients hemolysis under control if possible. Any transfused blood is basically incom-

patible since all adult red cells contain the I antigen. Many feel transfusion of cold blood is contraindicated. An efficient inline warmer is used.

Recent studies have shown some success in these patients employing plasmapheresis in the treatment of cold agglutinin disease. The anti-I molecule is an IgM immunoglobulin which is primarily intravascular and can be removed by plasmapheresis. This can provide temporary relief and make further transfusion therapy simpler.

The second case illustrates an autoimmune hemolytic anemia in a young woman caused by a warm type of autoantibody secondary to the underlying condition of systemic lupus erythematosus.

This 32-year-old multigravida meets many of the diagnostic criteria of systemic lupus erythematosus. Any four of 14 proposed criteria are diagnostic. This patient has the following 8 manifestations of systemic lupus erythematosus: 1) facial erythema with a classic butterfly rash; 2) discoid lupus erythematosus; 3) photosensitivity; 4) arthritis without deformity; 5) a positive LE preparation; 6) hemolytic anemia; 7) false-positive flocculation serologic test for syphilis); 8) pleuritis.

A mild normochromic, normocytic anemia is seen in 80% of patients with systemic lupus erythematosus. The more severe hemolytic anemia due to a warm autoantibody is seen in only 5% of the cases. This case illustrates hyperacute hemolysis during which the hematocrit may plunge as low as 5 vol/dl. In this case, the hemoglobin remains stable within normal limits for six years from the onset of her first symptoms of systemic lupus erythematosus. An upper respiratory infection and the stress of early pregnancy probably precipitated a severe hemolytic reaction which caused her hemoglobin to drop from 10.8 gm/dl to 3.5 gm/dl within a week. Pregnancy often increases the severity of hemolytic anemia. At the time of admission she showed signs of cardiovascular failure with shortness of breath and tachycardia.

Laboratory examination showed a profound normochromic, normocytic anemia with adequate production by the bone marrow as evidenced by an increased corrected reticulocyte count. The direct and indirect antiglobulin tests were positive indicating the presence of antibody both on the red cells and in the serum. Although the direct antiglobulin test is the most important test in establishing the diagnosis of autoimmune hemolytic anemia, it does not seem to be useful in monitoring treatment of the disease. Warm autoantibodies are usually of the IgG variety which act by attaching to the red cell and subsequently interact with phagocytic cells of the reticuloendothelial system, leading to phagocytosis and destruction of the red cell. Red cells escaping

from the spleen with damaged red cell membranes become spherocytic after their sequestration in that organ. During the first admission, the patient responded well to two units of packed red cells and a trial of corticosteroids.

Prednisone is the mainstay of treatment in warm autoimmune hemolytic anemia. The usual dose is from 60 to 100 mgs/day. This dose can be rapidly tapered according to patient response. Corticosteroids are felt to act by three different mechanisms. The first is by suppressing the serum immunoglobulin level and depressing the specific antibody production by direct lytic action on lymphocytes. A second mechanism of action may be due to the alteration of the antibody avidity for the antigen on the red cell surface. A third mechanism of action may be altered clearance of antibody coated cells by the reticuloendothelial system. Prednisone is effective in 80% of cases of autoimmune hemolytic anemia. The response may be poor in patients with a secondary type of hemolytic anemia such as this patient had.

Six weeks after a spontaneous abortion, the patient again developed severe anemia. The peripheral blood smear showed a macrocytic type of anemia which is often seen when many reticulocytes which are larger red cells than the older adult red cells, are present. Again, symptomatic relief occurred after four units of packed red cell transfusion and an increase in the prednisone dose.

During the next several months, the patient was relatively stable until she developed a respiratory tract infection. The hemoglobin dropped to 8.8 gm/dl and because of the recurrent anemia, a splenectomy was done. 30 to 40 % of patients with the warm antibody variety of autoimmune hemolytic anemia will require a splenectomy in order to obtain optimal control. Frequently, the patients will still require corticosteroids but at lower dosage to control the hemolysis postsplenectomy.

The patient's prednisone was decreased and the patient was discharged to be admitted 3 days later in shock with an increasing white count and diffuse infiltrates on chest x-ray. One can suggest a diagnosis of acute pneumonia. Arterial blood gases showed a pattern of metabolic acidosis and hypoxiemia. She probably died of overwhelming sepsis with a possible component of acute adrenal crisis.

Dr. Carol Dickson's Diagnoses

- 1. Cold Agglutinin Disease (Case I)**
- 2. Warm Antibody Induced Autoimmune Hemolytic Anemia, Secondary To Lupus Erythematosus With Mortality Due To Overwhelming Pneumonia (Case II)**

DR. BARLOW: The cases today were not diagnostic problems. However, they do illustrate several important points. First, as Dr. Dickson has nicely pointed out, they represent the two varieties of autoimmune hemolytic anemia caused by warm and cold antibodies. Secondly, they point out that both of these diseases may be extremely serious and lead to death. In the first patient, the immediate cause of death was an acute anterior myocardial infarction. There was an incidental finding of a Schwannoma of the distal esophagus. The patient had severe coronary heart disease and I think we would have to conclude that the severe anemia had a part to play in myocardial decompensation and the acute myocardial infarction.

The case of the young woman is even more interesting. There was an overwhelming diffuse bilateral pneumonia involving all lobes. The patient died of overwhelming sepsis. Gram stain and Dieterle stains for legionella pneumophila were equivocal but direct fluorescent antibody stains for legionella pneumophila were negative. The patient showed manifestations of disseminated intravascular coagulation with small thrombi seen within the kidneys, adrenals, heart and other organs. There was myocardial and adrenal hemorrhage and necrosis to show that organ damage was associated with the process. Certainly this patient had several reasons for overwhelming sepsis which included: 1) corticosteroid therapy; 2) recent splenectomy; and 3) systemic lupus erythematosus. A definite organism did not grow on postmortem routine bacteriological culture.

FINAL ANATOMIC DIAGNOSES

- 1. COLD AGGLUTININ DISEASE WITH ACUTE MYOCARDIAL INFARCTION SECONDARY TO SEVERE CORONARY HEART DISEASE (CASE I)**
- 2. OVERWHELMING DIFFUSE PNEUMONIA WITH DISSEMINATED INTRAVASCULAR COAGULATION AND SECONDARY MYOCARDIAL, RENAL CORTICAL, AND ADRENOCORTICAL INFARCTION DUE TO SYSTEMIC LUPUS ERYTHEMATOSUS AND AUTOIMMUNE WARM HEMOLYTIC ANEMIA.**

*DR. ROBERT MARSCHKE: Was there any thought of doing an open lung biopsy on this patient?

* Specialist in Oncology, Sioux Valley Hospital and Central Plains Clinic and Assistant Professor of Medicine, School of Medicine, University of South Dakota.

DR. BARLOW: The patient came in with overwhelming sepsis and died within a two day period. Consideration was being given to various diagnostic procedures but she died very quickly. I should also note that this case took place over 8 years ago and is simply presented because it illustrates a number of interesting points.

***DR. P. K. ASPAAS:** I had the patient with cold agglutinin disease who was referred in with the history as described. The acute hemolytic process that this patient had as the manifestation of cold agglutinin disease does not respond to steroids very well and does not respond to cytotoxic agents for several weeks. Therefore, we were hard pressed to treat this gentleman. I might note that as is typical in cold agglutinin disease, his antibody reacted not only at room temperature but at 37°C as well. We considered doing plasmaphoresis but his blood agglutinated even in warmed tubing. We did try steroids. The patient developed back pain the night before he expired and we were fearful that he was developing a myocardial infarction. We did transfuse him several times but to no avail before demise.

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Council Meeting Highlights

The Council of the South Dakota State Medical Association met on Saturday, November 22, 1980, at the Howard Johnson Motor Lodge, Sioux Falls, South Dakota. The following are major items of business transacted at this meeting.

1. **GOVERNMENT PROGRAM FEES FOR PHYSICIANS' SERVICES.** The Council passed a motion that the State Medical Association strongly agrees with the concept of accepting prevailing fees for the delivery of health care, and that if money allocated for Title 19 and Title 5 services has been depleted, the patients shall continue to have access to and receive essential care by physicians.

2. **DELIVERY OF HEALTH CARE BY THE HEALTH DEPARTMENT.** The Council took action to state that the State Medical Association supports the position that the South Dakota Department of Health be encouraged to refrain from involvement in health care delivery programs and be more appropriately concerned with traditional duties of the State Health Department.

3. **1981 LEGISLATIVE PROGRAM APPROVED TO DATE.**

- a) Introduce legislation to repeal the premarital serology testing law.
- b) Support limited sampling of prescription drugs at physicians' request.
- c) Endorse the Hospital Association's action to repeal the law which prohibits discrimination in county hospitals between practitioners of the various healing arts.
- d) Introduce legislation to repeal the law which requires each physician to record his license in the office of the Register of Deeds of the county where he resides and practices.

4. **AMA GUIDELINES FOR CATEGORIZATION OF HOSPITAL EMERGENCY CAPABILITIES.** The Council voted not to accept the AMA drafted guidelines because they are not applicable to South Dakota; however, if in the future a program is drafted that better suits South Dakota, the SDSMA would be interested in reviewing such a proposal.

5. **MEDICAL SCHOOL AND POSTGRADUATE PROGRAMS IN SOUTH DAKOTA.** The Council

adopted the following resolutions and directed they be sent to the Governor.

"The SDSMA strongly endorses the continuation of support for the School of Medicine and the existing residency programs and recommends assessment of other postgraduate disciplines as they become required for continuing accreditation of the Medical School by the LCME."

"The SDSMA will not prioritize the financial arrangements for residencies under the School of Medicine."

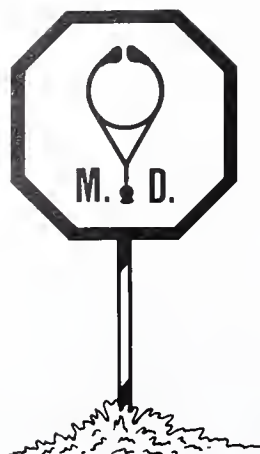
"The SDSMA reaffirms its support of the Medical School and its integral residency programs as a method for producing family practice oriented physicians for South Dakota."

6. **STATE HEALTH DEPARTMENT HYPERTENSION PROGRAM.** The SDSMA recommends that money be expended only for identification of people with hypertension problems in areas where pre-existing programs are not available, and then those identified be referred to a physician for care.

7. **STATE HEALTH DEPARTMENT FAMILY PLANNING PROGRAM.** The Council discussed the SDSMA's past experience in dealing with the family planning program and feels it has left a great deal to be desired. Some of the concerns expressed which have been relayed to representatives of the Health Department include:

- a) Inappropriate reimbursement levels.
- b) A contract which must be signed to participate in this program is most offensive to South Dakota physicians.
- c) The program appears to be unable to restrict its services to those persons who are in greatest need; i.e. the indigent. (The program will not refuse anyone its services irrespective of financial ability to pay.)

8. **HMO FEASIBILITY STUDY BY SOUTH CENTRAL COMMUNITY ACTION PROGRAM.** The Council directed the executive secretary to correspond with South Dakota's Congressional delegation expressing the concerns of the SDSMA regarding this HMO feasibility study and the money expended. ■



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The Medical Associates Clinic, Pierre, has announced the addition of **Raymond J. Owens, M.D.** to its staff as a pediatrician. Dr. Owens is a native of South Dakota. He received his BMS degree from the University of South Dakota Medical School in 1971 and his M.D. degree from Tufts University School of Medicine in Boston, Massachusetts in 1973. He completed his internship at Sioux Valley Hospital in Sioux Falls. Dr. Owens had a general practice in Pierre for 3 1/2 years before taking a pediatrics residency at the University of Tulsa Medical College. Dr. Owens and his wife Linda have two children.

* * * *

Robert Dappen, M.D. has opened his internal medicine practice in Mitchell. Dr. Dappen, who is a native of Lucas, received his M.D. degree from the University of South Dakota Medical School in 1977. He then completed three years of residency at the University of Oklahoma in Tulsa. Dr. Dappen and his wife, Cindy, a native of Mitchell, have one daughter.

* * * *

Raymundo Tan, M.D. has joined Radiology Services, P.A., Aberdeen. Dr. Tan came to this country from Manila, Philippines where he received his M.D. degree from the University of St. Tomas. He served his internship at the University of St. Tomas Hospital and the Cook County Hospital in Chicago, Illinois and completed his residency in radiology at Cook County Hospital in June, 1980. Dr. Tan and his wife Maria reside in Aberdeen.

* * * *

Sioux Valley Hospital, Sioux Falls, has named **Robert Henrickson, M.D.**, Sioux Falls, as the new director of the kidney dialysis unit. As director, Dr. Henrickson is in charge of both chronic dialysis and in- and out-patient care.

The Medical Clinic, Yankton, announces the association of **John Sternquist, M.D.** in the Department of General Surgery. Dr. Sternquist, a native of South Dakota, attended the University of South Dakota Medical School for two years and received his M.D. degree from the University of Minnesota. He took his residency in general surgery at the Hennipen County Medical Center. Dr. Sternquist and his wife Nancy, formerly from Beresford, have two children.

* * * *

Drs. Mike and Marsha Delaney have joined the Delaney Clinic, Mitchell. They are the first husband and wife team to practice in Mitchell and Dr. Marsha is the first woman general surgeon in the city.

Dr. Mike Delaney is a native of Mitchell. He received his B.S. degree from the University of South Dakota and his M.D. from the University of Missouri in Columbia. His first year residency was completed in general surgery and his second and third year residencies in otolaryngology.

Dr. Marsha Delaney received her M.D. degree from the University of Oklahoma. She interned in surgery at the University of Oklahoma also and served her surgical residency at Sacred Heart Hospital, Yankton and at the University of Missouri. In 1979 she received her board certification and practiced for two years in Columbia, Missouri before joining the Delaney Clinic.

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SD

Future Meetings

March

Internal Medicine Update, Sioux Valley Hosp., Sioux Falls, SD, March 4. 1 hr. AMA Category I credit. Contact: USD School of Med., McKennan Hosp., Sioux Falls, SD 57101. Phone: (605) 339-7573.

Neurology Lecture Series—Disorders of Sleep, V. A. Hosp., Ft. Meade, SD, March 5. 1 hr. AMA Category I credit. Contact: USD School of Med., McKennan Hosp., Sioux Falls, SD 57101. Phone: (605) 339-7573.

Neurology Lecture Series—Advanced Clinical Neurology, V. A. Hosp., Hot Springs, SD, March 6. 1 hr. AMA Category I credit. Contact: USD School of Med., McKennan Hosp., Sioux Falls, SD 57101. Phone: (605) 339-7573.

Pediatric Update, Sioux Valley Hosp., Sioux Falls, SD, March 11. 1 hr. AMA Category I credit. Contact: USD School of Med., McKennan Hosp., Sioux Falls, SD 57101. Phone: (605) 339-7573.

Advances in Internal Medicine West River Lectures—Public Health, V. A. Hosp., Ft. Meade, SD, March 12. 1 hr. AMA Category I credit. Contact: USD School of Med., McKennan Hosp., Sioux Falls, SD 57101. Phone: (605) 339-7573.

Neurology Lecture Series—Aphasias and Dysarthrias, Human Services Center, Yankton, SD, March 12. 1 hr. AMA Category I credit. Contact: USD School of Med., McKennan Hosp., Sioux Falls, SD 57101. Phone: (605) 339-7573.

Advances in Internal Medicine West River Lectures—Public Health, R. C. Regional Hospital, Rapid City, SD, March 13. 1 hr. AMA Category I credit. Contact: USD School of Med., McKennan Hosp., Sioux Falls, SD 57101. Phone: (605) 339-7573.

Advances in Internal Medicine West River Lectures—Public Health, V. A. Hosp., Hot Springs, SD, March 13. 1 hr. AMA Category I credit. Contact: USD School of Med., McKennan Hosp., Sioux Falls, SD 57101. Phone: (605) 339-7573.

Internal Medicine Update, Sioux Valley Hosp., Sioux Falls, SD, March 18. 1 hr. AMA Category I credit. Contact: USD School of Med., McKennan Hosp., Sioux Falls, SD 57101. Phone: (605) 339-7573.

Psychiatry Lecture Series—Affective Disorder, V. A. Hosp., Ft. Meade, SD, March 19. 1 hr. AMA Category I credit. Contact: USD School of Med., McKennan Hosp., Sioux Falls, SD 57101. Phone: (605) 339-7573.

Cardiac Ultrasonography, Memorial Hosp., Watertown, SD, March 19. 1 hr. AMA Category I credit. Contact: USD School of Med., McKennan Hosp., Sioux Falls, SD 57101. Phone: (605) 339-7573.

Therapy of Arrhythmias in Acute Myocardial Infarction, Huron Reg. Med. Ctr., Huron, SD, March 23. 1 hr. AMA Category I credit. Contact: USD School of Med., McKennan Hosp., Sioux Falls, SD 57101. Phone: (605) 339-7573.

Symposium on Gynecology, Oncology and Endocrinology, Yankton, SD, March 27-28. 8 hrs. AMA Category I credit. Contact: USD School of Med., McKennan Hosp., Sioux Falls, SD 57101. Phone: (605) 339-7573.

EKG Interpretation and Arrhythmia Management, Aladdin Hotel, Las Vegas, NV, March 27-29. 13 hrs. AAFP & AMA Category I credits. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-8646 toll free.

April

Thirty-Seventh Annual Congress of the American College of Allergies, Sheraton Washington Hotel, Washington, D.C., April 4-8. AMA Category I credits. Contact: Shirley Schoenberger, Exec. Sec., Am. Coll. of Allergists, 2141 Fourteenth St., Boulder, CO 80302. Phone: (303) 447-8111.

Coronary Heart Disease—1981, Hyatt Regency Milwaukee, Milwaukee, WI, April 9-11. 16 hrs. AAFP & AMA Category I credits. Fee: \$250. Contact: Mrs. Dorothy Black, Pub. Rel. Dept., St. Luke's Hosp., 2900 W. Oklahoma Ave., Milwaukee, WI 53215. Phone: (414) 647-6388.

Technology Assessment Forum on Coronary Artery Bypass Surgery: Economic, Ethical and Social Issues, Sheraton Washington Hotel, Washington, D.C., April 21-23. Contact: Elaine M. Kokiko, Exec. V.P., Moshman Assoc., Inc., 6400 Goldsboro Rd., Washington, D.C. 20034. Phone: (301) 229-3000.

Radiation Therapy Seminar, U. of Iowa, Iowa City, IA, April 23. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

May

Cardiac Rehabilitation, Hyatt Regency, Chicago, IL, May 1-2. 13 hrs. AMA Category I credits. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CA 80112. Phone: 800-525-8646.

Reproductive Endocrinology, U. of Iowa, Iowa City, IA, May 4-5. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

Cardiology Today, U. of Iowa, Iowa City, IA, May 4-8. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

Eleventh Annual Meeting of the Great Plains Organization for Perinatal Health Care, Radisson South, Bloomington, MN, May 14-16. Contact: Virginia Rittenour, Coordinator, Box 50, 420 Delaware St., S.E., Minneapolis, MN 55455. Phone: (612) 373-5718.

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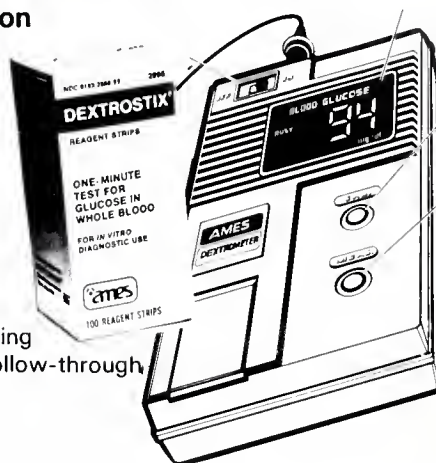
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SOUTH DAKOTA JOURNAL OF MEDICINE

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The University of South Dakota School of Medicine Alumni Association was officially organized in 1980. It is a self-standing, incorporated organization. It is responsible for alumni programming and raising of funds for general support of the School of Medicine. The Association works closely with the South Dakota Medical School Endowment Association which provides funds for student loans and scholarships and for research support. Both organizations are administered by separate Boards of Directors with assistance from the School of Medicine.

As of 1977 the University of South Dakota School of Medicine is a four-year degree granting school, and through the Alumni Association the school, and past and present students will be better served.

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Enzymatic Determination Of Serum Cholesterol And Triglycerides In Children Of South Dakota

Larry A. Lemaster, M.D.*
Thomas Aceto, Jr., M.D.*
Alfred E. Hartmann, M.D.*
Victor Morris, Ph.D.*

ABSTRACT

We have performed a pilot study to determine normal serum cholesterol and triglycerides levels on school aged children in this area by means of enzymatic determination. This sample population consisted of 162 school children enrolled in the Sioux Falls public school system, 1% of the total student population. The serum cholesterol, mean and standard deviation for males 5 to 11 years, was 169 ± 30 ; 12 to 18 years 159 ± 31 ; for females 5 to 11 years 180 ± 36 ; 12 to 18 years 165 ± 30 . The mean serum triglycerides level for males 5 to 11 years was 40 ± 10 ; 12 to 18 years 61 ± 35 ;

for females 5 to 11 years 69 ± 35 ; 12 to 18 years 66 ± 43 . We conclude that the fully enzymatic quantitation of cholesterol provides an accurate means of measuring cholesterol in the pediatric population that is both safer and quicker than the conventional extraction methods. The enzymatic method is also very useful for the screening of children. We feel that the enzymatic quantitation of triglycerides provides similar advantages in safety and speed and should be re-evaluated by additional studies in a comparable midwestern population.

Several large scale, well designed studies on cholesterol and triglycerides levels in normal children have been completed and have provided excellent data in determining the upper limits of normal.¹⁻³ In those studies, fasting cholesterol and triglycerides were quantitated colorimetrically following the extraction methods described in the combined Lipid Research Clinics Program and U. S. National Heart, Lung and Blood Institute study.⁴ Newer enzymatic

methods for determining cholesterol and triglyceride provide a number of advantages over the older methods. Our research was designed as a pilot study to determine normal serum cholesterol and triglycerides levels for school age children in our area by means of enzymatic determination.

MATERIALS AND METHODS

The sample population consisted of 162 school children enrolled in the Sioux Falls Public School System. The Sioux Falls Juvenile Diabetes Association provided support and helped organize drawing of the samples. Letters were sent to the parents of

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students in two elementary schools (grades 1 through 6); one junior high school (grades 7 through 9), and one high school (grades 10 through 12), asking permission to draw blood samples from their healthy children. We requested the subjects to fast following supper the night before venipuncture. Students who were taking any medications, or whose parents did not consent to the study, were excluded from our sample. Five per cent of the students in the four schools were studied. This consisted of one per cent of the total students in the Sioux Falls School system. The sample population consisted entirely of white children, reflecting the essentially all white population of the city.

Venipunctures were performed between 8:00 a.m. and 11:00 a.m. during the months of October through December. Each child was questioned immediately before venipuncture to verify overnight fasting. Sera were kept frozen until all the sample collections could be completed.

Analysis for serum cholesterol and triglycerides was performed using fully enzymatic methods. The method used to quantitate cholesterol was developed by Roeschlau, based upon the combined use of cholesterol esterase and cholesterol oxidase resulting in a highly specific, totally enzymatic analysis. The cholesterol esterase hydrolyzes the cholesterol esters to cholesterol and free fatty acids. The cholesterol is oxidized to 4-cholesten-3-one, with the production of hydrogen peroxide. The hydrogen peroxide reacts with 4-aminoantipyrine and phenol when catalyzed by peroxidase to form a colored quinoneimine which is then measured colorimetrically.^{5,6} The method used to quantitate triglycerides was based on Wahlefeld's procedure, except that liver esterase was replaced with esterase from a microorganism, facilitating fast and complete lipolysis at 20-25°C.⁷⁻⁸ Coefficients of variation for normal cholesterol and triglycerides samples produced day to day precision of 2.1 and 1.5 per cent respectively.

Table I							
Serum Cholesterol and Triglycerides Levels							
Cholesterol							
Males				Females			
Age (Yrs.)	n.	Mean (mg/dl)	S. D. (mg/dl)	Age (Yrs.)	n.	Mean (mg/dl)	S. D. (mg/dl)
5-11	15	169	30	5-11	18	180	36
12-18	66	158	31	12-18	63	165	30
Triglycerides							
5-11	15	40	10	5-11	18	69	35
12-18	66	61	35	12-18	63	66	43

RESULTS

Table I displays the number, mean, and standard deviation for serum cholesterol and triglycerides by sex when divided into age groups 5-11 years and 12-18 years. This table was calculated following the exclusion of all values that exceeded two Standard Deviations for that sex and age group. These age groups were chosen for two reasons. First, it facilitates ease of comparison with the Cincinnati study. Secondly, the dramatic increase with age in triglyceride levels as demonstrated in previous studies occurs at about 12 years of age.¹

DISCUSSION

Enzymatic vs. Conventional Methods.

Enzyme linked analysis of cholesterol and triglycerides provides significant advantages over the conventional extraction methods. The enzymatic method not only saves labor and time, but negates the need for large quantities of organic solvents which are both dangerous and difficult to handle. In addition, the enzymatic methods are more conducive to automation, thereby providing an efficient means for determining cholesterol and triglycerides in larger numbers. Such automation would also be very useful in screening children for hyperlipoproteinemias.

Allain et al compared the enzymatic determination of serum cholesterol with those obtained by the classical Liebermann-Burchard procedure and found that the enzymatic method was reproducible and provided better specificity than the conventional method. Results of the study also showed high correlation when comparing the two methods on 97 different samples.⁶

Earlier studies comparing determination of serum triglycerides by use of enzymes versus use of the fluorometric Hantzsch procedure showed excellent results. Results of 30 assays in each of three serum pools of triglycerides demonstrated reproducibility. Assays of 38 different serum samples for triglycerides by auto-analyzer and enzyme methods showed very similar results for both methods. Plotting the enzymatic method versus the auto analyzer procedure produced a correlation coefficient of 0.993 and a linear regression of $y = 1.048x - 4$.

The Bogalusa and Cincinnati studies used the conventional extraction methods (Lipid Research Clinic, Auto-Analyzer II) to quantitate cholesterol and triglycerides levels.⁴ There was general agreement of the Sioux Falls study with the Bogalusa Heart Study and the University of Cincinnati lipid study (Table II).

Statistical evaluation by use of t values, comparing Sioux Falls males to Bogalusa males, and

Table II
Comparison of Three Different Serum Lipid Studies
Serum Cholesterol

Caucasian Males

Caucasian Females

	Age (Yrs.)	n	Mean (mg/dl)	S. D. (mg/dl)		Age (Yrs.)	n	Mean (mg/dl)	S. D. (mg/dl)
Bogalusa ⁽²⁾	5-14	1142	161.3	28.7		5-14	1030	163.5	27.1
Sioux Falls	5-18	81	159	33		5-18	81	169	31
*Cincinnati ⁽¹⁾	6-11	1359	159			6-11	1200	162	
	12-17	1243	153			12-17	1144	155	

Serum Triglycerides

+Bogalusa ⁽²⁾	5-14	1033	68.9	33.5		5-14	976	77.3	37.6
Sioux Falls ⁺	5-18	81	57	33		5-18	81	67	41
*Cincinnati ⁽¹⁾	6-11	1359	51			6-11	1200	58	
	12-17	1243	71			12-17	1144	73	

* plasma
+ serum

Sioux Falls females to Bogalusa females (as displayed in Table II) enables us to assume equivalent means for total male and total female serum cholesterol ($t = .069$ and $t = 1.74$ respectively.) However, comparing the same groups for total male and total female serum triglycerides results in t values of 3.08 and 2.35 respectively. Therefore, we are not able to assume equivalent means for triglycerides levels.

We have no definite explanation why the serum triglycerides levels differ. It has been shown that serum triglycerides is age related.¹ However, the Sioux Falls group being older than the Bogalusa group, yet having lower levels of serum triglycerides for both males and females, is the opposite of what one would expect. Perhaps our small sample population, a relatively cool season, climate, and a different diet could have affected our study.

In conclusion, our study demonstrates that the fully enzymatic quantitation of cholesterol provides an accurate means of measuring cholesterol in the pediatric population that is both safer and quicker than the conventional extraction methods. Also, the fully enzymatic method is a very useful method for screen of children.

We feel that the enzymatic quantitation of triglycerides provides similar advantages in safety and speed and should be re-evaluated by additional studies on a comparable midwestern population.

We wish to give special thanks to Miss Sonja Starks and Mrs. Eileen Hyatt, technologists; Mrs. John Craney, Sioux Falls Juvenile Diabetes Foundation; and Mrs. Mary Lerssen, R.N., Sioux Falls Public School system for their assistance.

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A CENTENNIAL NOTE—

In 1907 only 45 percent of the physicians in South Dakota were members of the State Association. In hopes of increasing the membership to 500-600, the Association hired a member, Dr. J. L. Stewart, to travel the state and get new members. He was given an expense account of \$300. In his first report to the Association Dr. Stewart boasted 73 new members; however, he failed to collect the dues at the time he signed them up, thus his efforts were not totally successful. In 1914-1915 the AMA agreed to send an organizer to South Dakota to solicit new members if the State Association would pay \$1.00 for each new member gained.

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bocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K⁺ frequently; both can cause K⁺ retention and elevated serum K⁺. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Anti-hypertensive effect may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with

possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia, although uncommon, has been reported. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function.

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—Thomas Edison
Inventor

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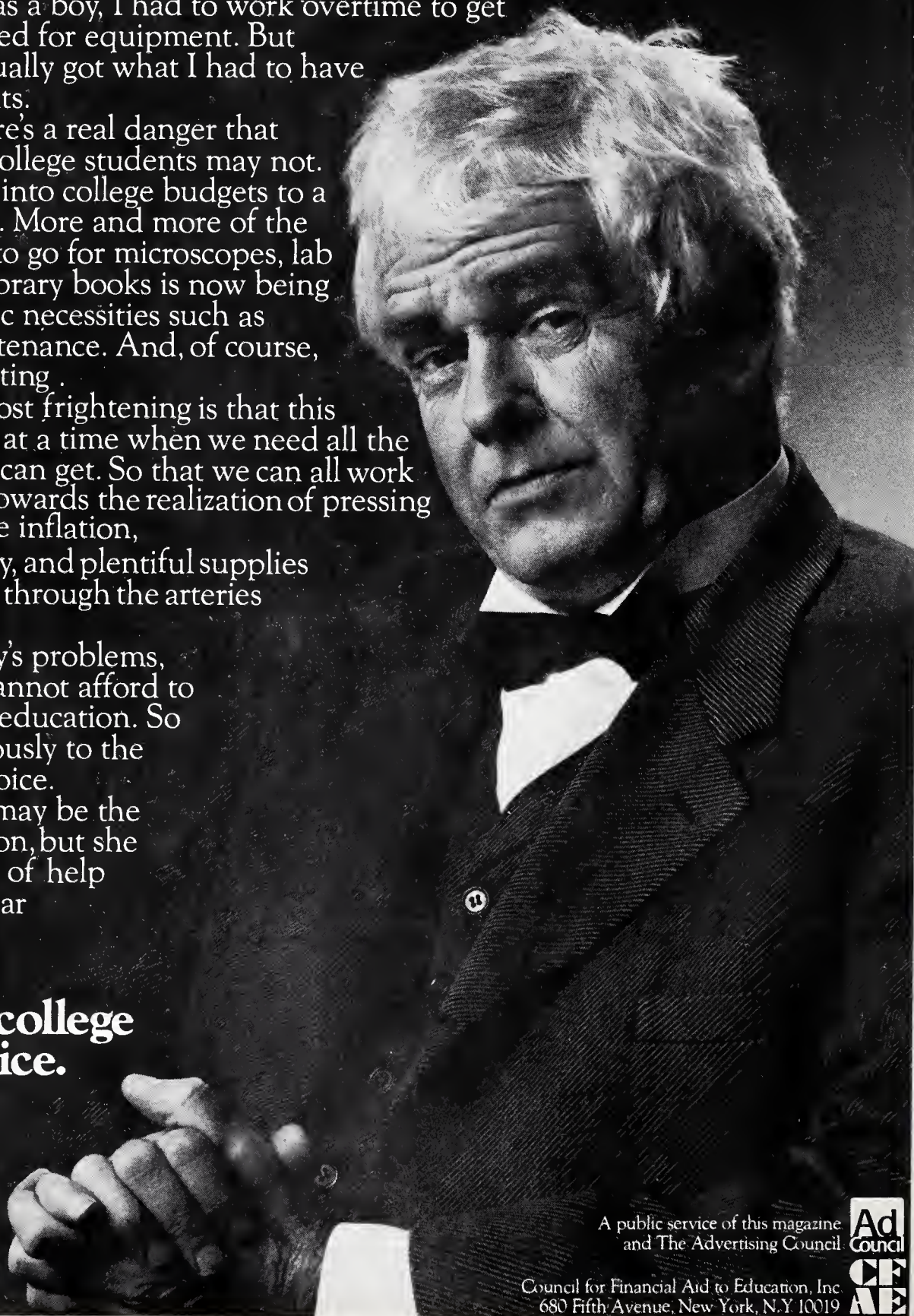
Today there's a real danger that many American college students may not. Inflation is eating into college budgets to a dangerous degree. More and more of the money that used to go for microscopes, lab equipment and library books is now being consumed by basic necessities such as heating and maintenance. And, of course, my specialty—lighting.

What is most frightening is that this squeeze is coming at a time when we need all the trained minds we can get. So that we can all work more effectively towards the realization of pressing goals: manageable inflation, revitalized industry, and plentiful supplies of energy coursing through the arteries of this country.

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Twenty-One Year Old Primigravida With Recurrent Left Flank Pain And Anemia

John Jones, M.D.*
Discusser

John F. Barlow, M.D.**
Editor

Case No. 862 286

This 21-year-old primigravida entered Sioux Valley Hospital with a chief complaint of left flank pain.

The patient first presented with flank pain at 4½ months gestation. The pregnancy had otherwise been normal without morning sickness, proteinuria, or hypertension. The flank pain was on the left and started suddenly for no apparent reason. It was a sharp pain that radiated down the left flank. It was relieved by lying down to some degree but mild analgesics did not seem to relieve the pain. There was no associated nausea, vomiting or urinary symptoms. The patient was not febrile.

The patient had had trouble with burning in the epigastric area since high school. This was not relieved by food but was relieved by antacids which she had taken periodically for this pain since the time of onset. She had had no hematemesis or melena and she never had had x-rays of the gastrointestinal tract. There was a family history of peptic ulcer disease. The patient had had a tonsillectomy without difficulty at age 3. She had had post-streptococcal glomerulonephritis at age 7.

PHYSICAL EXAMINATION: Pulse: 100/min and regular; Respirations: 20/min and regular; Temperature: 98.6°F; Blood pressure: 110 systolic and 66 diastolic; Weight: 141 lbs. Examination of the head and neck was unremarkable. The lungs were clear to auscultation and percussion. The heart was not enlarged. There was a grade 1 systolic ejection type murmur heard

along the left sternal border without radiation. Examination of the abdomen revealed an intruterine pregnancy measuring 13 cm. with fetal heart tones at 150/min. There was pain in the left upper quadrant on deep palpation as well as in the left flank below the costal margin. Pelvic examination revealed no masses or abnormality other than the pregnant state. Neurological examination was unremarkable.

LABORATORY DATA: Urinalysis: yellow-hazy, specific gravity 1.002 pH 6.0, negative for protein, glucose, trace ketone bodies, negative for protein, bile, and hemoglobin; sediment negative. Hemoglobin 9.4 gm/dl, hematocrit 28 Vol/dl with normal blood cell indices. Total leukocyte count 8,900/mm³ with 58% segmented neutrophils, 17% neutrophilic bands, 2% eosinophils, 23% lymphocytes. The red cells were normochromic and normocytic. The platelets were normal in number and morphology on the smear. A direct antiglobulin was within normal limits. A serum iron was 22 ug/dl (normal 50-180 ug/dl), iron binding capacity 329 ug/dl (normal 200-400 ug/dl), percent saturation 7% (normal 20-44%). Haptoglobin was within normal limits. A serum ferritin was 120 ng/ml (normal 10-155 ng/ml). Lactic dehydrogenase (LDH), alkaline phosphatase, aspartate aminotransferase (SGOT), total bilirubin, total protein, calcium, phosphorus, glucose, blood urea nitrogen, creatinine, uric acid, and cholesterol were within normal limits. A creatinine clearance was 178 ml/min. An electrocardiogram was unremarkable. An intravenous pyelogram (IVP) showed a left flank soft tissue density of undetermined etiology at the lower pole of the kidney but probably not representing spleen and possible extrarenal. There was a minimal left basilar infiltrate compatible with pneumonia. An ultrasound study revealed a single fetus with a somewhat transverse lie and a biparietal diameter corresponding to 19 weeks gestation. The placenta was posterior without evidence

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of abruption or low implantation. Left kidney was not well visualized and there was a suggestion of fluid collection behind this kidney. The spleen could not be visualized. The patient was transfused with 4 units of packed cells with a rise in hematocrit from 24% to 35%. The patient was thought to have a retroperitoneal hematoma of unknown etiology.

The patient did well for 3 months, but then developed another episode of sudden onset of left flank pain. A firm 25 cm. tender area in the left flank below the 12th rib was noted. The hematocrit had dropped from 34 vol/dl to 30 vol/dl within two weeks. Physical examination was unremarkable, except for 32 weeks gestation and the area of tenderness and firmness to palpation in the left flank. Urinalysis was unremarkable. Hemoglobin 10.3 gm/dl, hematocrit 30 vol/dl with normal indices, total leukocyte count 11,500/mm³ with 80% segmented neutrophils, 7% neutrophilic bands, 10% lymphocytes, and 3% monocytes. Platelet count 290,000/mm³. Prothrombin time 11.9 seconds with a control of 11.5 seconds. Partial thromboplastin time 27.8 seconds, with a control of 28.0 seconds. A direct antiglobulin test and haptoglobin were negative. An ultrasound showed a single intrauterine fetus. No hematoma was noted. The hemoglobin dropped to 9 gm/dl. The patient was transfused with 2 units of packed cells which brought the hemoglobin to a height of 11.5 gm/dl. It was 10.6 gm/dl on discharge. She was discharged on the 5th hospital day.

One week after discharge, the patient again developed recurring knife-like pain in her left flank which was even worse than the pain she had previously. This did not seem to radiate and was not relieved by any particular maneuver. Physical examination was unchanged. Admission urinalysis was again negative. Hemoglobin was 10.5 gm/dl. A limited computer tomography of the abdomen revealed a mass involving the left kidney. An intravenous pyelogram revealed an 11 cm. in diameter mass lesion over the lower pole of the left kidney with areas of hematoma within it. An aminocentesis showed a total lecithin sphingomyelin (L/S) ratio total was 3.2/1.0, acetone precipitate (L/S) was 1.9/1.0. No phosphatidyl glycerol was present. These studies indicated borderline fetal pulmonary maturity. The patient was again discharged and readmitted at term after an L/S ratio indicated maturity. An operation was performed.

DR. JONES: This 21-year-old white pregnant female presented with left flank pain. Pain in the left flank may arise from structures in the left side of the abdomen including the kidney, ureter, spleen, large bowel, or stomach. Flank pain could also be referred from disease in sites such as the lung or pelvis. The fact that the urine is normal and the white blood count is not elevated would tend to exclude infection in spite of the frequency of urinary tract infections in pregnancy. There was a mild left shift in the white count and the hemoglobin was low. These findings can be associated with stress or bleeding. A low hemoglobin is often seen in pregnancy due to inadequate iron intake and normal hemodilution in pregnancy. This was supported by the low serum iron with a normal total iron binding capacity and a low percent saturation. The negative direct antiglobulin test indicated that there was not autohemolysis causing the anemia. The chemistry panel indicated that renal and hepatic functions were normal as well as a normal metabolic state. The creatinine clearance of 178 ml/min is normal for a pregnant female because of the increased glomerular filtration rate associated with pregnancy. She had an ab-

dominal ultrasound which demonstrated the fetus and suggested a collection of fluid around the kidney. A chest x-ray showed a left basilar infiltrate which could have indicated pneumonia which could be a cause of referred pain to the left flank from pleural irritation. More likely the left abdominal pain caused atelectasis of the left lower lung resembling an infiltrate. An intravenous pyelogram (IVP) was done showing a left flank soft tissue density possibly extrarenal. At this point the hematocrit dropped from 28 vol/dl to 24 vol/dl during the hospitalization and she was transfused with 4 units of blood. The rapid development of anemia, the sudden onset of left flank pain, and the appearance of a left flank mass suggested that she had a tumor or cyst with bleeding into it resulting in a sudden increase in the size of the mass with pain due to stretching of the peritoneum. The most common mass would be a simple renal cyst especially with an otherwise normal IVP. A cyst would be 5 to 8 times more common than a renal cell carcinoma in a young female. The second episode of pain was also associated with a rapid drop in the hematocrit indicating the pain was due to a rapid expansion of the mass secondary to hemorrhage. An IVP with the second episode showed an 11 cm. soft tissue mass deforming the lower pole of the kidney. The mass was obviously involving the kidney at this time. A limited CT scan of the abdomen at the level of the renal mass showed variable density within the mass suggesting at least blood and fat. Masses from the kidney that contain fat are unusual. The most common soft tissue mass in the kidney is a hypernephroma (adenocarcinoma, renal cell carcinoma). This type of tumor does not contain fat and should give a homogenous mass as should all of the various types of malignant processes. The most common benign soft tissue tumor would be an angiomyolipoma or hamartoma. These lesions are often associated with tuberous sclerosis but may arise in the absence of this entity.

These tumors can be present in three ways: 1) a unilateral single tumor without the signs of tuberous sclerosis; 2) multiple and bilateral hamartomas with widespread connective tissue defects of tuberous sclerosis; or 3) a fetal hamartoma. The CT scan of an angiomyolipoma may be diagnostic if there is a significant amount of fat within the mass. This patient had no evidence of tuberous sclerosis and she was an adult so the diagnosis is almost certainly angiomyolipoma. If this is the diagnosis, treatment would consist of nephrectomy or possible selective renal artery embolization.

Dr. John Jones' Diagnosis:
Angiomyolipoma Of Kidney With Hemorrhage

DR. BARLOW: Dr. Hoskins, will you show the imaging studies:

*DR. HOSKINS: The first significant study was the intravenous pyelogram in which one can see a mass distorting the lower portion of the collecting system and involving a large area about the lower pole of the left kidney (Figure 1). The fetus is demonstrated and one can see the hydronephrosis and hydroureter in the opposite collecting system typical of the changes seen in pregnancy.



Figure 1

Intravenous pyelogram showing large mass occupying lower pole of left kidney. There is dilatation of the left collecting system as seen in pregnancy. Fetal skeleton can be seen.

The CT scan shows the tumor at the lower pole of the kidney. This is an atypical picture of angiomyolipoma of the kidney because one cannot see the typical fat densities seen in this lesion. The hemorrhage has probably obscured the characteristic pattern (Figure 2).

Our urology group has seen five cases of angiomyolipoma of the kidney in the past 12 years—3 women and 2 men. Many of the cases demonstrated some of the more characteristic x-ray appearances

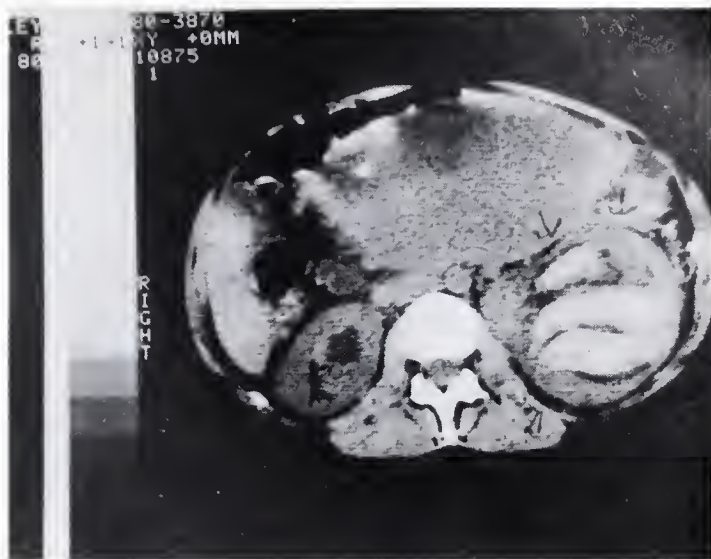


Figure 2

CT scan showing hemorrhage into left renal mass (arrows).

with the fat densities in the kidney. Often only intravenous pyelograms with nephrotomograms are necessary to make a presumptive diagnosis. On one patient, because we suspected a renal cyst, a needle biopsy was attempted and resulted in a bloody tap.

An arteriogram is often helpful but the differential diagnosis between a renal cell carcinoma and angiomyolipoma can be extremely difficult. Neither tumor can be differentiated by epinephrine injection.

One woman presented with a massive hemorrhage and shock secondary to rupture of the lesion. Two of the men had smaller renal lesions and had nephrectomies with preoperative diagnoses of renal cell carcinoma. However, the pathologic diagnosis was angiomyolipoma. The tumor is supposed to be more common in women and slightly more common on the left than on the right.

DR. BARLOW: Material from the larger lesion was submitted for frozen section. The first sections did not reveal typical changes of angiomyolipoma but the second specimen revealed the trinity of tissues typical of this lesions—blood vessels, adipose tissue and smooth muscle. The gross photograph reveals a large lesion in the lower pole and a second smaller lesion in the upper pole (Figure 3). The upper pole lesion measures 8.5 cms. in greatest dimension and the lower pole lesion 4.5 cms. Cut sections of both lesions shows abundant central hemorrhage. Microscopic sections again show the three different types of tissue referred to above in varying proportions (Figure 4). The vessels can vary from capillary to almost arterial-like in nature. Thrombosis in these blood vessels or rupture because of lack of tissue support can explain the frequent presentation of these lesions as massive hemorrhage. Occasionally,

* Urologist, Sioux Valley Hospital, Sioux Falls, SD; and Head, Section of Urology, School of Medicine, University of South Dakota.

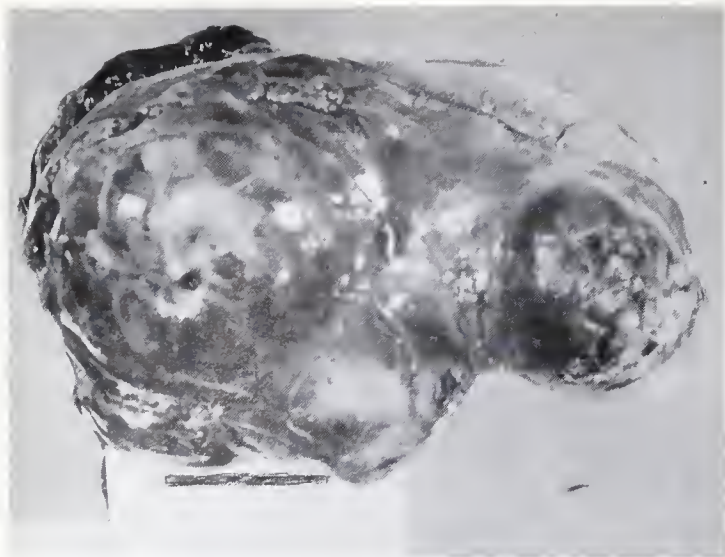


Figure 3

Gross photograph revealing two tumors with hemorrhage at opposite poles of kidney.



Figure 4

Photomicrograph demonstrates vascular, adipose and smooth muscle elements of angiomyolipoma. H & E 100X.

the smooth muscle components can show some pleomorphism of the nuclei which can lead to misinterpretation of a malignancy of the kidney.

Angiomyolipoma or hamartoma of the kidney were first described in 1800 by Bourneville and Brissand. They appear in 50 to 80% of cases of tuberous sclerosis and are often multiple and bilateral in this condition. They occasionally can be large and asymptomatic. The sex incidence is equal in this autosomal dominant condition. Angiomyolipomas can also be seen in the absence of tuberous sclerosis. There is a female predominance. According to Price and Mostofi, the average age of occurrence is

approximately 41 years, but the age range is from 12 to 69 years. The major manifestation of the lesion is, as in this case, hemorrhage. The patient may present with sudden onset of abdominal pain, hematuria, syncope, nausea, vomiting, tenderness and flank mass. This may occur suddenly with shock or may be intermittent over a period of time, such as in this patient. Some cases have had mild or nonspecific symptoms or even present as fever of unknown origin.

FINAL ANATOMIC DIAGNOSIS ANGIOMYOLIPOMA OF THE KIDNEY (ONE AT EACH POLE) WITH HEMORRHAGE

*DR. W. WITTMAN: Was a needle biopsy of this lesion considered?

**DR. D. G. ORTMEIER: No, needle biopsy was not attempted because the patient was thought to have a retroperitoneal hematoma and it was feared further hemorrhage might occur.

***DR. JERRY SIELEFF: I would imagine further x-rays and more definitive CT scan was not attempted on this woman because of fear of x-ray during pregnancy. How dangerous is this?

DR. JONES: The most dangerous time for x-rays during pregnancy is during the first three months.

DR. HOSKINS: It should be noted that x-rays taken at term may be the worst time in regard to an adverse effect on future fertility for the fetus.

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A CENTENNIAL NOTE—

In 1886 the DAKOTA MEDICAL BRIEF was established as the Dakota Medical Society's official publication. Drs. Sevey and Andros of Mitchell were in charge of the first publication. The association was beginning to be noticed and accepted—the rail service even gave members reduced rates when traveling to and from conventions.

* Resident in Family and Community Medicine, Sioux Valley Hospital, Sioux Falls, SD.

** Family Practitioner, Sioux Valley Hospital, Sioux Falls, SD, and Clinical Faculty of School of Medicine, University of South Dakota.

*** Resident in Family and Community Medicine, Sioux Valley Hospital, Sioux Falls, SD.



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This product is prepared from human venous plasma. Each individual unit of plasma has been found nonreactive for hepatitis B surface antigen using the radioimmunoassay method of counter-electrophoresis.

INDICATIONS

Treatment of rabies, once clinical disease becomes apparent, is rarely if ever successful. Rabies vaccine (duck-embryo origin, Lilly Laboratories) with or without Rabies Immune Globulin (Human)—Hyperab[®] should, therefore, be given to all persons suspected of exposure to rabies, particularly to severe exposure. Whenever possible, Hyperab[®] globulin should be injected as promptly as possible after exposure. If initiation of treatment is delayed for any reason, however, Rabies Immune Globulin (Human) should be given just the same, regardless of the interval between exposure and treatment.

Rabies virus is usually transmitted by the bite of a rabid animal, but can occasionally penetrate abraded skin with the saliva of infected animals. Progress of the virus after exposure is believed to follow a neural pathway, and the time between exposure and clinical rabies is a function of the proximity of the bite (or abrasion) to the central nervous system and the dose of virus injected. The incubation is usually 2 to 6 weeks, but can be longer. After severe bites about the head and neck, it may be as short as 10 days.

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AAFP Resident Activities Fact Sheet

Continued to support family practice residency graduates in their efforts to obtain hospital privileges for which they are qualified and trained . . .

Continued to advocate a system for documentation of both the quantity and quality of family practice residents' hospital experience . . .

Achieved success with JCAH in gaining revisions in its "Accreditation Manual," which will permit departments of family practice to be organized and operated in the same manner as other departments . . .

Expanded liaison with the Joint Commission on Accreditation of Hospitals by attaining representation on two JCAH special committees: the Hospital Accreditation Program Professional and Technical Advisory Committee and the Ambulatory Health Care Program Professional and Technical Advisory Committee . . .

Continued to provide workshops in the areas of practice management and coping with practice pressures with special workshops geared toward residents training in family practice . . .

Distributed sets of 32 videotaped "Vignettes," which portray several types of stressful situations family practice residents might experience, to all approved family practice residency programs free of charge . . .

Established a research program in health care delivery, a research panel for clinical research and, began development of a bibliography of family practice research . . .

Began developing protocols to study the cost-effectiveness of delivery of medical care by family physicians . . .

Continued representation of family physicians before various government committees and agencies on such matters as insurance, FDA regulations, funding for family practice residency programs and physician reimbursement under Medicare and Medicaid, etc. . . .

Produced the Resident Organization Guide, designed to assist family practice residents interested in developing organized resident groups . . .

Cooperated with the American Board of Family Practice and the Society of Teachers of Family Medicine on the development of a prototype in-training assessment program . . .

Continued sponsorship of the annual National Conference of Family Practice Residents designed for resident affiliate members of AAFP to increase their involvement in Academy activities . . .

Continued the bi-monthly "AAFP Resident and Student Newsletter" in the Academy's monthly **AAFP Reporter** . . .

Expanded resident representation (with voting privileges) to national Academy commissions and committees . . .

Appointed voting delegates to Academy's Congress of Delegates, thereby providing residents direct input into overall policy-making decisions of AAFP . . .

Continued to provide residents easy access to the AAFP headquarters office via the Academy's toll-free number—800-821-2512 . . .

Launched an intensive membership recruitment campaign geared toward women training in family practice residency programs to solicit their input and involvement in Academy programming . . .

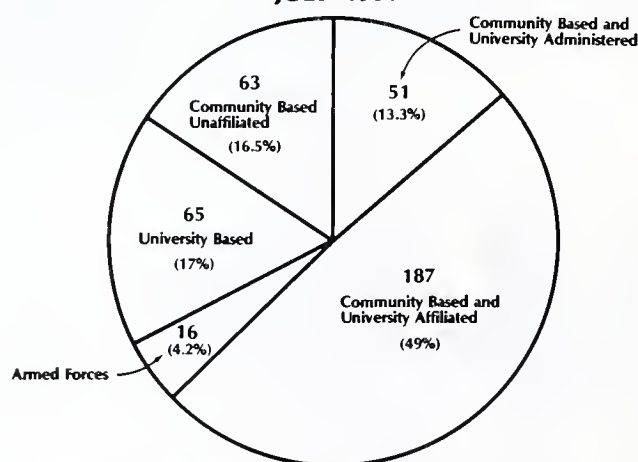
Began periodic site visits to family practice residency programs for the purpose of increasing resident membership in AAFP, as well as to provide residents an opportunity to request information on Academy activities relating to residents in family practice. (These one on one visits have had a significant bearing on increasing resident membership.) . . .

Continued to waive registration fee for residents attending the AAFP Annual Scientific Assembly, which also provides a resident lounge (for informal conferences during the meeting) and a reception for residents attending the Convention and,

Established a Committee on Minority Health Affairs which has direct input from physicians training in family practice.

For additional information on any of the above programs, call AAFP today—toll-free (800-821-2512).

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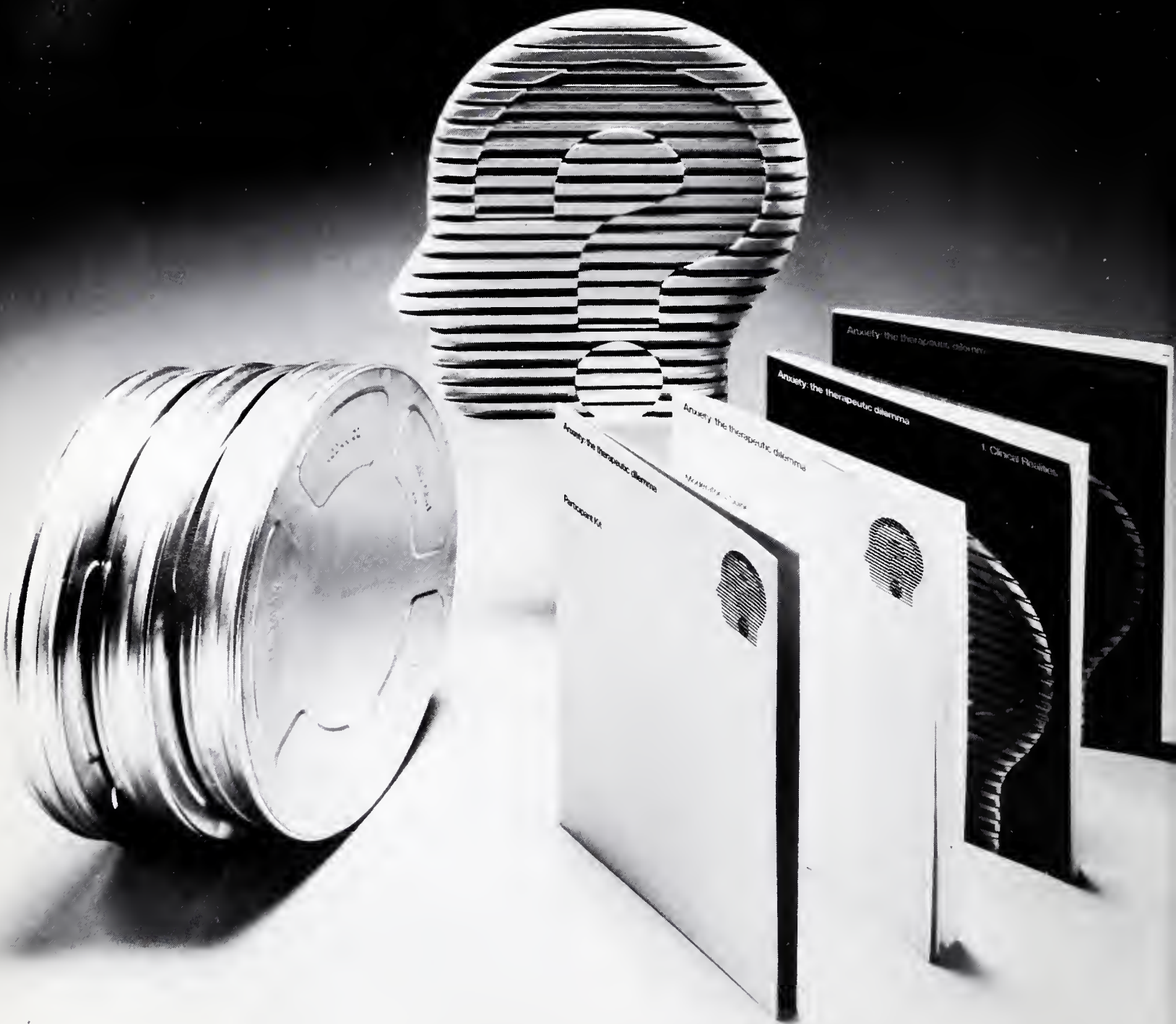
New Bylaws

SDAFP members attending the Annual Business Meeting in Rapid City, August 14, 1981, will be asked to vote on a revised set of Bylaws. This will conform to changes made by AAFP, deleting the Constitution and incorporating it into uniform Bylaws suggested for constituent (state) chapters. A copy of the proposed Bylaws is on file at the state office for members, upon request.

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From Beltline To Steering Wheel: The Vanishing Space

An Informal Study of Persistent Weight Gain of Truck Drivers*

Richard J. Rather, P.A.-C.
Sherry Warriner, R.D.
Darrell Johnson, R.N.

ABSTRACT

Obesity, particularly in truckers, was the target of an informal study by the Industrial Medicine Department, Central Plains Clinic, Ltd., Sioux Falls, SD.

On-the-road eating and lack of activity are major factors in creeping weight gain. Some ideas for off-setting the gain trend are offered. Walking,

even just around a parking lot or the truck, is one easy-to-follow bit of advice. Several methods of cutting calories while adding nutrition are suggested, i.e. carrying a cooler for fruits, vegetables, lo-cal drinks, ordering a la carte, utilizing salad bars. Regular balance of eating, driving and sleeping is also detailed, along with specific examples of lo-cal meals, beverages, etc.

A study of obesity in truck drivers was recently undertaken by the Industrial Medicine staff and registered dietitian of Central Plains Clinic, Ltd., of Sioux Falls, SD. Being aware of widespread generalized weight gain in truckers and the difficulties encountered in losing, CPC personnel also set forth some simple methods of curtailing those gains.

A common definition of obesity is an excessive

accumulation of body fat¹—indeed, 30% of men and 40% of women, both between the ages of 40 and 49, are more than 20% above “ideal” weights established by Metropolitan Life Insurance. Interestingly, 10-15% of young children and 30% of adolescents are obese. Many truckers, particularly those who drive over the long hauls, face slow, gradual weight gains which persistently close the gap between belt and steering wheel.

Causes, definitions and specific ways to fight the “battle of the bulge” are outlined below.

When caloric intake exceeds caloric expenditure, obesity is the result. For most of us, the high consumption of calories begins to slack off after the late

* Under the auspices of: CENTRAL PLAINS CLINIC, LTD., 2727 S. Kiwanis Avenue, Sioux Falls, SD 57105. Phone: 605-335-2727. Michael R. Ferrell, M.D., Medical Director. Department of Industrial Medicine, Edward H. Peters, M.D., Guy E. Tam, M.D.

teens, when caloric needs are only for body tissue maintenance, rather than for development of new tissue. Therefore, as we age, we need fewer calories for body fuel. The change is so subtle that some of us don't recognize what's happening until 10 lb. have crept on!

Another contributing factor in creeping weight gain is low activity level. The very job that provides a trucker a living for his family also presents a possible "diabolic duo" to his health—very low activity level (particularly on long hauls) and high caloric intake with meals on the road. In a snowballing effect, the resultant increased body weight may contribute to increased fatigue, high blood pressure, abnormal blood sugars, impaired pulmonary function and, most importantly, coronary heart disease. In addition, psychological problems may be triggered by obesity.

Our entire culture, of course, is preoccupied with obesity. Diets, exercise programs, "get-thin" groups and testimonials abound—check out your favorite magazine store, newspaper, bookstores; there are scores of plans to follow. "Obesity therapy" ranges from counting calories to behavior modification, with hundreds of stops in between; most physicians agree some behavior modification is necessary.

The informal gathering of information by the CPC Industrial Medicine staff and nutritionist lends rather graphic credence to a general statement pointing out that long-term and/or long-distance truck drivers *do* rather consistently gain weight. In a random sampling of drivers for Midwest Coast Transport of Sioux Falls, SD, done over a two-year period, the following statistics rapidly evolve:

- 82.5% GAINED an average of 11.91 lb.
- 12.5% LOST an average of 6.8 lb.
- 5.0% had NO CHANGE in weight.

One individual, George, at age 25 weighed 155 lb. After five years of trucking, he weighed 185 lb., and after ten years, he weighed 196 lb.

Additionally, the following four examples of change in weight by truckers are rather typical.

Trucker	Height	1978 Weight	1980 Weight
Driver 1	5'10"	217 lb.	227 lb.
Driver 2	6'	278 lb.	280 lb.
Driver 3	5'9½"	190 lb.	198 lb.
Driver 4	6'	222¼ lb.	235 lb.

The "battle lines" we have drawn will outline some ideas to minimize caloric intake, particularly while on the road, in addition to some simple behavioral pattern changes.

Loading and unloading a truck is heavy exercise;

however, the long hauls provide only pedal pushing. Every time you stop the truck (for refueling, meal time, rest stop, etc.), walk around the parking lot or even just around your truck several times. Your circulation will benefit, even though weight reduction may not be achieved, by distributing your body's fuel more efficiently. Of course, the best form of exercise is to push away from the table and leave the area. In doing so, you resist calories in the remaining foods at which you might pick if you were to stay there. Try to get some exercise at least once a day. Exercise is best if it is moderate but sustained, such as in walking or swimming.

Schedules are important in trucking; schedules are very important in eating also. When your trucking schedule is irregular, start your first meal of the day when you get up. By consuming some food about every five hours, people tend to be less hungry than if there are longer periods of time between feedings. The goal is to divide your food among the waking hours, keeping in mind that eating may not be a full meal, but a snack. We suggest the following schedule as an example:

10 p.m.	3 a.m.	8 a.m.	1 p.m.	6 p.m.	11 p.m.
Eat		Eat	Eat	Eat	
Drive→	Sleep→	Drive→	Sleep→	Drive→	Sleep

The total calories required per day are fairly low for truckers because of inactivity. Depending on age, body size and activity, 1500-1800 calories per day may be adequate for body maintenance. If these calories are spread out in the pattern described (every five hours), appetites are more easily controlled. A distinct hazard of overeating is drowsiness. When a large meal is consumed, the blood supply to the brain is reduced, due to its diversion to aid the digestive system. The reduced blood supply may cause a slowdown of brain activity which, in turn, can produce sleepiness. The dangers inherent here can lead to accidents. The CPC Industrial Medicine staff has learned that at least one death in a trucker is attributed, indirectly, to just such drowsiness. The particular overweight trucker pulled over to the side of the road to take a quick nap. He just bent over and rested his head on the steering wheel. While sleeping, he suffocated because his fat tissue pushed up against his lungs, and gradually, there wasn't enough air getting into the lungs to provide the oxygen for circulation to the vital body organs. He was later discovered dead, slumped over his steering wheel.

One important thing to remember is that skipping meals IS NOT saving calories. Eating three times daily is important, as it:

- 1) gives the body increased circulation,
- 2) improves body efficiency,

3) provides refueling as the body works.

We have some ideas for coping with restaurant meals, as listed below.

- 1) Breakfast: try to order single serving meals, for example, one egg, one slice toast, one serving of bacon or sausage. A la carte ordering may be the simplest way to get a smaller breakfast.
- 2) Lunch (or any light meal): try to order side dishes of vegetables or dinner salad to accompany a sandwich (rather than french fries or potato chips). Salad bars are good opportunities for adding fruits and vegetables, such as lettuce, carrot and celery sticks, bean salads, pickled beets, plain mixed fruits without dressings. Add only one spoonful of dressing to your salad.
- 3) Dinner (or any large meal): try to select small meat items. Meats are high in fat; each ounce of meat is about 75 calories. Choose between crackers and bread (about five squares of cracker equal one slice of bread). Choose one potato item per meal and try to control the amount of fat added, such as potato salad from the salad bar OR a baked potato with one pat of butter or one spoonful of sour cream OR hash browns.

Keep in mind that most restaurant meals are fairly high in fat. Whenever possible, select the lower calorie broiled or baked foods. In addition, practice not adding extra fats when you eat the foods. Salt can retain fluids and in doing so, can aggravate and elevate blood pressure. Restaurant foods may well be salted in cooking; therefore, eliminating habitual salting at the table will help limit salt intake. Steak sauces, canned or packaged hams and bacon are also high in salt content.

We also offer, just as general information that we hope will be helpful to truckers, a few more ideas and important facts. Water is an essential nutrient. It is recommended that adults consume 4-8 cups of water a day. Water is an excellent no-calorie replacement for sweetened beverages, such as soft drinks. Tea is a source of caffeine, is best taken unsweetened or artificially sweetened. Filling a large thermos with ice and tea may be a good substitute for soft drinks during hot weather.

Choosing fresh or dried fruits or canned juices in place of candy or sweetened beverages will save calories and provide some essential nutrients.

Some fairly low-caloried munchies are Veri-Thin pretzel sticks, and unbuttered, only slightly salted (if at all) popcorn. Try eating one stick or one kernel at a time—and enjoy every single chew! Sunflower seeds and nuts, though nutritious, have fairly high caloric value in the form of fat.

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See "WARNINGS"

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Candy bars and soft drinks can be included occasionally, but when used as meal replacements, they provide only empty calories.

Some truckers find it helpful to start out a run with a cooler filled with fresh fruits, raw vegetables, sandwiches and beverages. (Carrying fruits which don't spoil easily, such as apples and oranges, is possible even without a cooler.) On the return trip, they eat at cafes. The combination of meal plans helps control calories and the cost of eating. There are many sizes of coolers and/or thermos jugs from which to choose to fit your space limitations.

If you share driving with a partner, consider sharing meals, too, making overeating much more difficult. You might, for example, split a sandwich and then each order a salad to round out the meal.

In summary, as a general rule, truckers gain weight from inactivity and restaurant meals that are too large and contain too many calories, as well as, often, too few food groups. Some possible ways to alter this trend include ordering smaller meals, trying to not add extra calories with fat and empty-calorie snacks, cutting back on salt and increasing the fruit and vegetables in the diet. In addition, try for a time/meal balance, such as every-five-hour-eating. Lastly, but perhaps most importantly, develop some regular means of exercise. Then watch the space between your belt and your steering wheel get larger while you enjoy a new sense of well-being.

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A CENTENNIAL NOTE—

In 1916 the State Public Health Association was proposed with its primary purpose to education. The feeling being that what was needed was an intelligent public sentiment which will insist that as citizens and taxpayers the state shall use a reasonable amount of care in protecting them against disease. The Association's Committee on Health and Public Instruction reported, "We have the most inefficient and underpaid Department of Public Health in America. The sum of \$8,000 is the total amount available for all purposes, while I am told, the stallions of the state get \$16,000 and the fish and game over \$40,000."

S
D

This Is Your Medical Association

Alton Ochsner, M.D., world renowned surgeon, medical pioneer and native of South Dakota, has established a \$10,000 endowed scholarship at Dakota Wesleyan University for graduates of Kimball High School. Dr. Ochsner, a 1914 graduate of Kimball, created the fund to honor this year's Centennial Celebration of Kimball. The earnings from the permanent fund will assure scholarship assistance yearly to Kimball students at Dakota Wesleyan. Dr. Ochsner received his medical education from the University of South Dakota and Washington University in St. Louis, Missouri and also additional studies in German and Switzerland. He now resides in New Orleans, Louisiana where he founded the Ochsner Clinic and Hospital.

* * * *

Aberdeen obstetrician, **Kumud Shinghal, M.D.**, has been elected a Fellow of the American College of Obstetricians and Gynecologists. She has practiced in Aberdeen since 1972.

* * * *

A Rapid City physician, **Norman, D. Neu, M.D.**, has been named to a three-year term as vice chairman of the South Dakota section of the American College of Obstetricians and Gynecologists.

* * * *

Curtis M. Adams, M.D. has joined the Yankton Clinic, Yankton. Dr. Adams received his medical degree at the U. of Minnesota in 1976. Prior to coming to the Yankton Clinic, he completed his residency in obstetrics and gynecology at Sacred Heart Hospital, Yankton. Dr. Adams and his wife, Brenda, have two children.

* * * *

John Barlow, M.D., Pathologist, Lab. of Clinical Medicine, Sioux Falls, has passed the American Board of Pathology examination for subspecialty certification in the field of Microbiology. Dr. Barlow has been previously certified in anatomic and clinical pathology and by the American Board of Nuclear Medicine.

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Acute Lupus Erythematosus (SLE) Following Polyvalent Pneumococcal Vaccine

Kristen Ries, M.D., F.A.C.P.*
Natalie K. Shemonsky, M.D., F.A.C.P.**

ABSTRACT

A patient is presented in whom an acute lupus erythematosus syndrome with pericarditis developed following vaccination with pneumococcal vaccine (Pneumovax).

Pneumococcal vaccine has been available for two years. Its role, safety and efficacy is still being questioned.^{1,2} Since the release of the vaccine, there have been many reports of mild febrile reactions associated with pain, redness and swelling at the site of the injection.^{1,3} Reports of more serious and long lasting systemic febrile reactions are few in number; however, morbidity was marked in these cases.^{4,5,6} Kippel et.al. reported successful immunization of patients with SLE without untoward effects.⁷ This report concerns the apparent precipitation of an acute systemic lupus erythematosus episode following vaccination with pneumococcal vaccine.

A sixty-four year old woman who was previously diagnosed to have hypothyroidism, hypertension, osteoarthritis, and diverticulosis was maintained on triamterene-hydrochlorthiazide (Dyazide) and levothyroid (Synthroid). On October 18, 1978 she received 0.5 ml of pneumococcal vaccine (Pneumovax). She did not return until eight days later when

she reported an illness which began approximately ten hours after the vaccine was administered. Her first symptoms included right-sided pleuritic chest pain, fever and chills. These symptoms continued unabated over the entire eight days only varying as to the side of the chest which was involved.

Physical examination revealed a blood pressure of 102/80, pulse 112 per minute, temperature 38.9°C(R). She was pale, uncomfortable with chest pain and appeared much older than previously. There was no neck vein distention. Examination of the lungs revealed crepitant rales heard especially over the right base posteriorly. The heart sounds were now distant, but no rub was appreciated. There was no Kussmaul sign.

Laboratory studies revealed a hemoglobin of 9.2 mg% (down from her previous 12 mg%); fluorescent antinuclear antibody (FANA) positive in a titer of 1:1280; sedimentation rate (Westergren) 118 mm/hour; WBC 11,300 with 78 segs, 2 bands, 13 lymphs, and 7 monocytes; Coombs test negative; LE preparation positive.

Chest X-ray revealed considerable increase in cardiac size compared to a previous film and was suggestive of pericardial effusion. Echocardiogram per-

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formed at a nearby medical center was consistent with pericardial effusion.

Within twenty-four hours of the institution of aspirin and bedrest, she began to improve. Thus, no further therapy was instituted in light of her clinical improvement and normal complement levels. She was asymptomatic within two weeks and her cardiac size was normal one month later. She remains asymptomatic as of this time.

Two years ago a new polyvalent pneumococcal vaccine (Pneumovax) was released for general use by the medical profession. The vaccine was intended for the prevention of infection due to the fourteen most usual types of *S. pneumoniae*. The recommendations for use of the vaccine vary from very loose (e.g. anyone over the age of fifty or in a chronic care facility) to very narrow (e.g. patients who have sickle cell anemia or who have had splenectomy). Contraindications include only children less than age two, pregnant women, and hypersensitivity to any of the components of the vaccine.³

Idiopathic systemic lupus erythematosus (SLE) is a clinical syndrome associated with the formation of autoantibodies and generalized tissue injury which is mediated by immune complexes. It has been recognized that certain events can precipitate or exacerbate SLE (e.g. drugs, vaccines, infections).⁷

Steinberg suggests that SLE is a multifactoral disease in which a genetic predisposition combined with environmental stimuli are necessary for disease expression. These stimuli vary with different individuals. A strong enough environmental stimulus may induce disease in an individual with the genetic trait for SLE.⁸

Autoantibodies, especially rheumatoid factor, have also been found after immunization of humans with viral preparations and other vaccines.^{7,8} Thus in SLE, certain viruses or protein products previously tolerated could lead to an immune response causing disease. Pneumovax may be one of many exogenous agents which could precipitate SLE.

Our patient received the vaccine because of her age and underlying chronic diseases. The vaccination was followed within ten hours with chills, fever and pleuritic chest pain which continued over eight days. At that time she was found to have pleuropericardial chest pain, anemia, pericardial effusion by chest X-ray and echocardiogram, a positive FANA in a titer of 1:1280 as well as a positive LE preparation. She had no evidence of cardiac tamponade and as she was already improving on supportive therapy alone, no further therapy was instituted.

In summary, we have presented a patient who developed an acute episode of SLE following vaccination with pneumococcal vaccine. This raises the question of the use of this vaccine in patients with

lupus erythematosus despite reports suggesting safety in these patients. This disease should be considered in the evaluation of any patient with a significant reaction to the pneumococcal vaccine.

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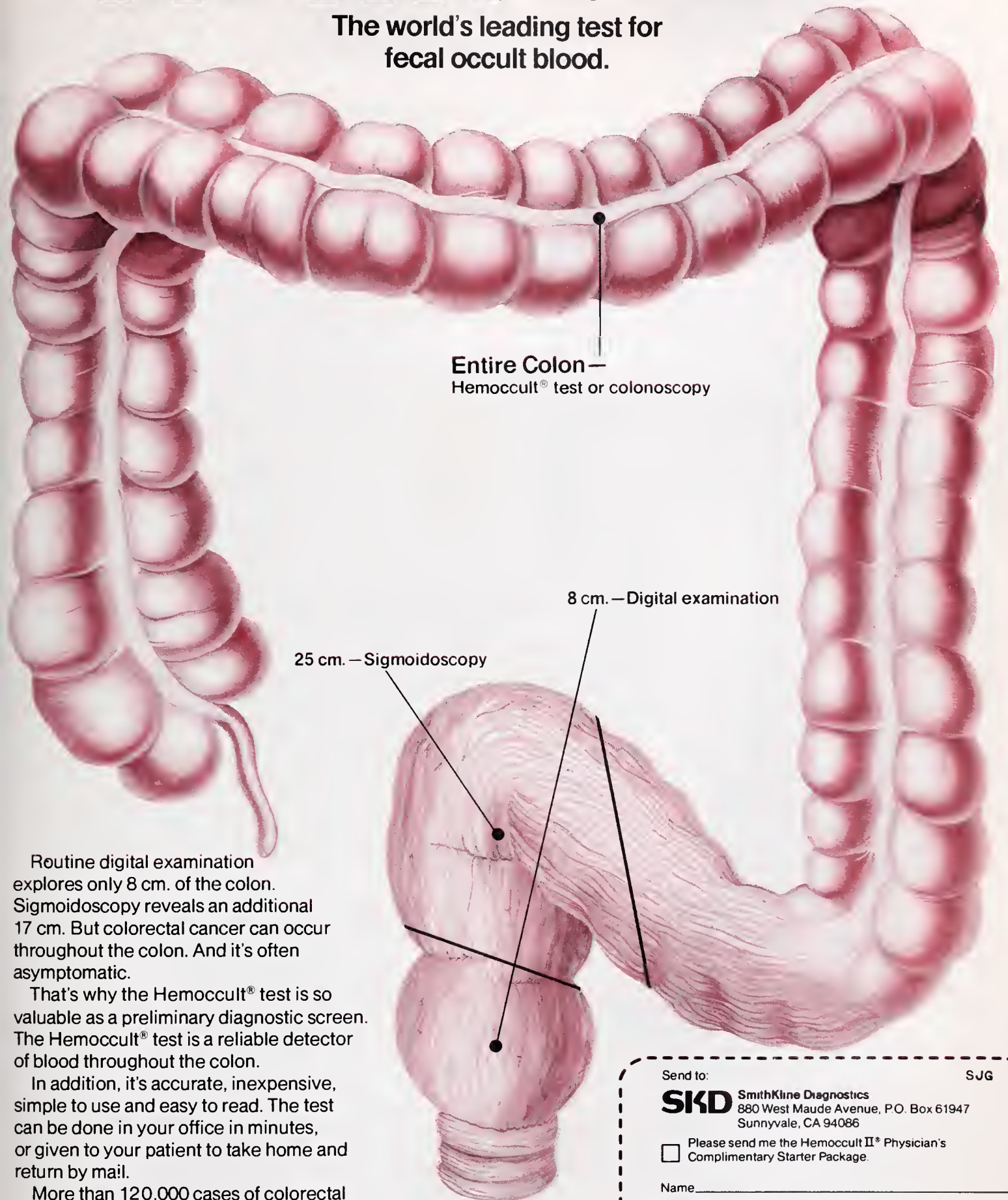
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Use in ambulatory patients: Empirin with Codeine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. The patient using this drug should be cautioned accordingly.

Interaction with other central nervous system (CNS) depressants: Patients receiving other narcotic analgesics, general anesthetics, phenothiazines, other tranquilizers, sedative-hypnotics, or other CNS depressants (including alcohol) concomitantly with Empirin with Codeine may exhibit an additive CNS depression. When such combined therapy is contemplated, the dose of one or both agents should be reduced.

Use in pregnancy: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. Therefore, Empirin with Codeine should not be used in pregnant women unless, in the judgment of the physician, the potential benefits outweigh the possible hazards.

PRECAUTIONS:

Head injury and increased intracranial pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a pre-existing increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.

Acute abdominal conditions: The administration of Empirin with Codeine or other narcotics may obscure the diagnosis or clinical course in patients with acute abdominal conditions.

Allergic: Precautions should be taken in administering salicylates to persons with known allergies: patients with nasal polyps are more likely to be hypersensitive to aspirin.

Special risk patients: Empirin with Codeine should be given with caution to certain patients such as the elderly or debilitated, and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture, peptic ulcer, or coagulation disorders.

ADVERSE REACTIONS: The most frequently observed adverse reactions to codeine include light-headedness, dizziness, sedation, nausea and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include euphoria, dysphoria, constipation, and pruritus.

The most frequently observed reactions to aspirin include headache, vertigo, ringing in the ears, mental confusion, drowsiness, sweating, thirst, nausea, and vomiting. Occasional patients experience gastric irritation and bleeding with aspirin. Some patients are unable to take salicylates without developing nausea and vomiting. Hypersensitivity may be manifested by a skin rash or even an anaphylactic reaction. With these exceptions, most of the side effects occur after repeated administration of large doses.

DOSEAGE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient. It may occasionally be necessary to exceed the usual dosage recommended below in cases of more severe pain or in those patients who have become tolerant to the analgesic effect of narcotics. Empirin with Codeine is given orally. The usual adult dose for Empirin with Codeine No. 2 and No. 3 is one or two tablets every four hours as required. The usual adult dose for Empirin with Codeine No. 4 is one tablet every four hours as required.

DRUG INTERACTIONS: The CNS depressant effects of Empirin with Codeine may be additive with that of other CNS depressants. See WARNINGS.



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Due Process

Here is a subject which must be regarded with considerable urgency, because of the frequency with which procedure within the medical jurisdiction often leads to a break-down in subsequent legal investigations and court presentations. Due Process is that carefully stipulated and accepted procedure through which medical care or professional behavior come to investigation, by the physicians.

Most of the medical staff members of the various hospitals across the state are well aware of the importance of due process incorporating the timing, the legal documents, the committees, the appeals etc. Unfortunately, every aspect of critical review of our professional work must come under some form of due process or we cannot go far in either peer review or correction of known deficiencies. The development of due process, with relation to medical events, is a painful one; first requiring development of bylaws and regulations necessary to form the framework, and secondly applying without deviation, this framework to the solution of problems as they occur. Emotionalism and economic considerations often lead to departure from the due process in such a way that subsequent litigation will show the defects.

PSRO organizations, carrying the function of peer review for units of medical care not qualified or desirous of carrying on their own review, serve the need for the purposes of medicare. There is an area of need in review with correct due process necessary for all patient care circumstances. I am not referring here to federally funded programs or to federally regulated peer review. More important to all of us is that we are under great pressure to provide a fair review system, which has the due process built in.


The functions of the Grievance Committee are general and for the most part would function best in screening the problems sent to them for patients so that an equitable solution can be forthcoming. Many times this is in relation to the nonprofessional portion of medical care, that is fees and their collection or other personal aspects of the care delivered. These can be in reference to the aesthetics involved, the patient's impression of professionalism, the persistence in satisfying the individual rights of recipi-

ents of care. It would be my impression that the primary function of the Grievance Committee in this area would be to see that in the review of a situation that the problem is placed in the proper slot for correct evaluation. If the problem could truly be solved by an explanatory letter or interview with the patient this would suffice; however, if professional investigation of the problem is necessary it must be in due process through the proper channels. Fortunately most areas of medicine are covered by such a due process in the conduct of review by committees of the medical staff. We must develop a generally attested and accepted due process for any areas which are devoid of such.

It is a painful experience for physicians to try to deal with problems which may be serious and perhaps damaging to individuals when they are not justly directed by a firm due process system in managing these. I hope to bring before the Council of the Medical Association a critical review based on this discussion, not with the idea of adding to our already overburdened regulation, but to make sure that what we do on behalf of ourselves and our patients is not a waste of time for the people involved. I am sure there are many of you who have served on ad hoc committees or on the Grievance Committee or even the Board of Medical Examiners who have seen the results of malfunctions in this vast developing and adhered to due process system.

Many areas of the country have engaged in seminars and presentations for the benefit of members of the profession to develop a keen understanding of due process. We should, at our earliest convenience, do the same.

Sincerely yours,



Winston B. Odland, M.D., President
South Dakota State Medical Association

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Internal Medicine Update, Sioux Valley Hosp., Sioux Falls, SD, April 1. Category I credits. Contact: USDSM, McKennan Hosp. Sioux Falls, SD 57101.

Neurology Lecture Series—Advanced Clinical Neurology, V. A. Hosp., Ft. Meade, SD, April 2. Category I credits. Contact: USDSM, McKennan Hosp., Sioux Falls, SD 57101.

Neurology Lecture Series—Advanced Clinical Neurology, V. A. Hospital, Hot Springs, SD, April 3. Category I credits. Contact: USDSM, McKennan Hosp., Sioux Falls, SD 57101.

New Cardiac Drug Modalities, Sturgis, SD, April 8. Category I credits. Contact: USDSM, McKennan Hosp., Sioux Falls, SD 57101.

Advances in Internal Medicine West River Lectures—Infectious Diseases, V. A. Hospital, Ft. Meade, SD, April 9. Category I credits. USDSM, McKennan Hosp., Sioux Falls, SD 57101.

Neurology Lecture Series—Head Trauma, Human Service Center, Yankton, SD, April 9. Category I credits. Contact: USDSM, McKennan Hosp., Sioux Falls, SD 57101.

Advances in Internal Medicine West River Lectures—Infectious Diseases, V. A. Hospital, Hot Springs, SD, April 10. Category I credits. Contact: USDSM, McKennan Hosp., Sioux Falls, SD 57101.

The Challenge of Stress, Spearfish, SD, April 14. Category I credits. Contact: USDSM, McKennan Hosp., Sioux Falls, SD 57101.

Pediatric Ophthalmology and Strabismus, Sheraton-Ritz Hotel, Minneapolis, MN, April 13-14. Fee: \$180. 14 hrs. Category I credits. Contact: CME, Box 293 Mayo Memorial Bldg., 420 Delaware St., S.E., Minneapolis, MN 55455. Phone: (612) 373-8012.

Technology Assessment Forum on Coronary Artery Bypass Surgery: Economic, Ethical and Social Issues, Sheraton Washington Hotel, Washington, D.C., April 21-23. Contact: Elaine M. Kokiko, Exec. V.P., Moshman Assoc., Inc., 6400 Goldsboro Rd., Washington, D.C. 20034. Phone: (301) 229-3000.

Neurogenic Bladder and Spinal Cord Injury, Mayo Mem. Aud., U. of Minn., Minneapolis, MN, April 22-24. Fee: \$230. 20 hrs. Category I credits. Contact: CME, Box 293 Mayo Memorial

Bldg., 420 Delaware St., S.E., Minneapolis, MN 55455. Phone: (612) 373-8012.

Radiation Therapy Seminar, U. of Iowa, Iowa City, IA, April 23. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

Annual Allergy/Immunology Course, Mayo Mem. Foundation, U. of Minn., Minneapolis, MN, April 30-May 2. Fee: \$200. 17 AAFP & AMA Category I credits. Contact: CME, Box 293 Mayo Memorial Bldg., 420 Delaware St., S.E., Minneapolis, MN 55455. Phone: (612) 373-8012.

May

Cardiac Rehabilitation, Hyatt Regency, Chicago, IL, May 1-2. 13 hrs. AMA Category I credits. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CA 80112. Phone: 800-525-8646.

Reproductive Endocrinology, U. of Iowa, Iowa City, IA, May 4-5. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa Coll. of Med., Iowa City, IA 52242.

Cardiology Today, Univ. of Iowa, Iowa City, IA, May 4-8. Fee: \$275. 30 hrs. AAFP & AMA Category I credits. Contact: Carl W. White, M.D., Cardiovascular Div., Univ. of Iowa Hosp. & Clinics, Iowa City, IA 52242. Phone: (319) 356-2881.

Eleventh Annual Meeting of the Great Plains Organization for Perinatal Health Care, Radisson South, Bloomington, MN, May 14-16. Contact: Virginia Rittenour, Coordinator, Box 50, 420 Delaware St., S.E., Minneapolis, MN 55455. Phone: (612) 373-5718.

Proper Prescribing: Conflicting Signals, Mt. Sinai Sch. of Med., New York, NY, May 18-19. Category I credits. Fee: \$110. Contact: John P. Morgan, Sophie Davis School of Biomedical Ed., City Coll. of NY, Convent Ave. at 138th St., New York, NY 10031.

Otolaryngology Clinical Conference, Univ. of Iowa, Iowa City, IA, May 22. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa Coll. of Med., Iowa City, IA 52242.

June

John Lawrence Interdisciplinary Symposium on the Physical and Biomedical Sciences, Sioux Falls, SD, June 3-4. Contact: George P. Scott, Dept. of Chemistry, Univ. of SD, Vermillion, SD 57069.

Bariatric Surgery Workshop, Univ. of Iowa, Iowa City, IA, June 4-5. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa Coll. of Med., Iowa City, IA 52242.

Intensive Course in Pediatric Nutrition, Univ. of Iowa, Iowa City, IA, June 15-19. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa Coll. of Med., Iowa City, IA 52242.

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Some people feel that patients being treated with anxiolytic drugs are "weak," can't tolerate the anxieties of normal daily living, and should be able to resolve their problems on their own without the help of medication.

The FACT is that while most people can withstand normal, everyday anxieties, some people experience excessive and persistent levels of anxiety due to personal or clinical problems. An extensive national survey concluded that Americans who do use tranquilizers have substantial

Facts

justification as evidenced by their high levels of anxiety. It was further noted that antianxiety drugs are not usually prescribed for trivial, transient emotional problems.

Some people feel afraid of me because of the stories they've heard about my being harmful and having the potential to produce physical dependence.

The FACT is that there are thousands of references in the medical literature documenting my efficacy and safety. Extensive and painstakingly thorough studies of toxicological data conclude that I am one of the safest types of psychotropic drugs available. Moreover, I do not cause physical dependence if the recommended dosage and therapeutic regimen are followed under careful physician supervision. However, I can produce dependence if patients do not follow their physicians' directions and take me for prolonged periods, at dosages that exceed the therapeutic range. Patients for whom I have been prescribed should be cautious about their use of alcohol because an additive effect may result.

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Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety, symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy)

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Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

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Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other anti-depressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy.

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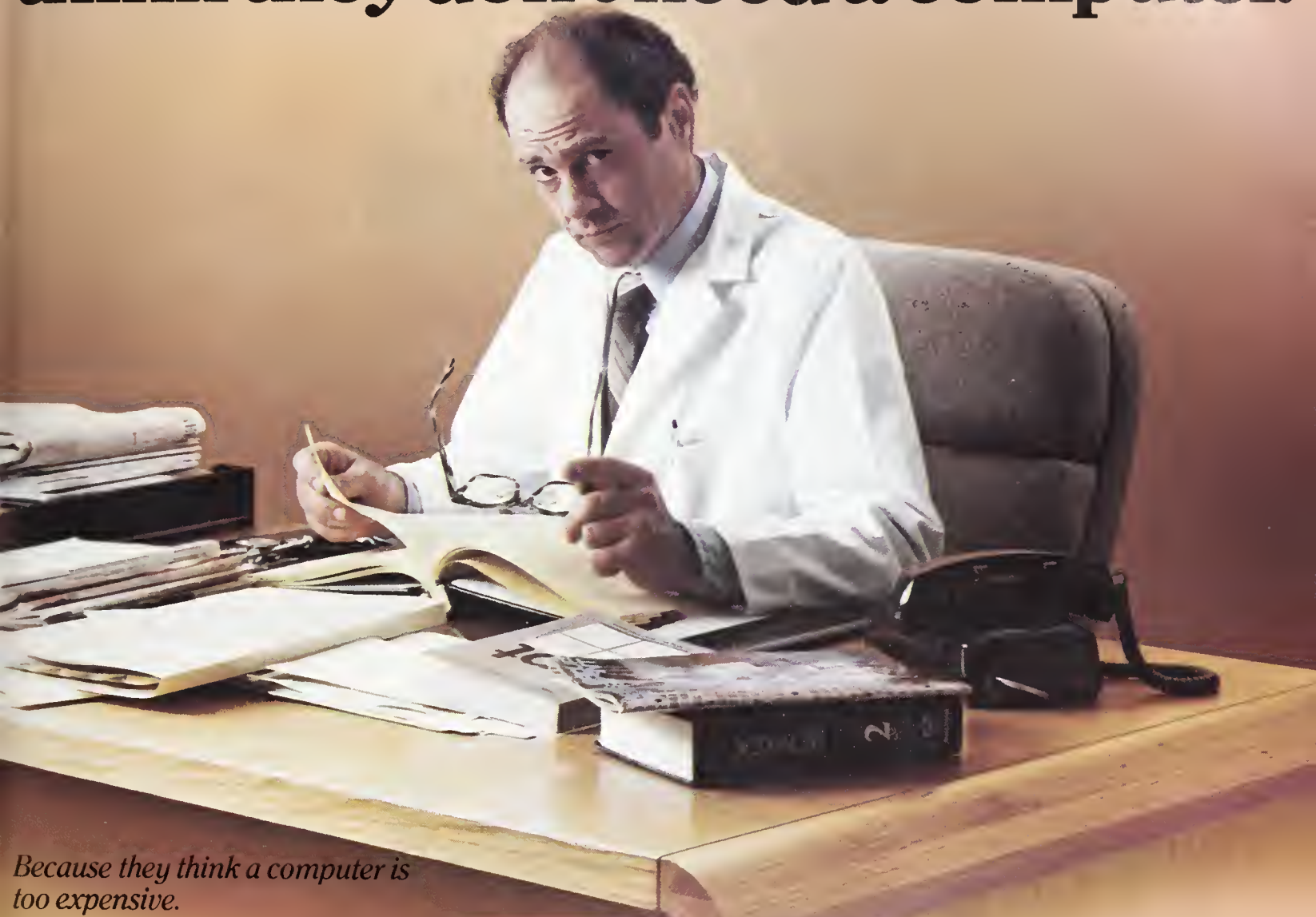
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SD

President's Page

Identity Crises—Not Really.

Reshuffling terminology gives comfort to new generations, adding a sense of creativity to their sometimes deficient lives. At times these identities can serve a cause for a short period lending importance and increased self-image if not true credibility or genuine service. Let me give you a few examples. An intern was once readily identified by all as a physician in training and perhaps described as "on the job". He was a graduate M.D. in most training hospitals, although he was kept humble by a variety of factors including constant supervision, correction and economic depression. Now we have interns in pharmacy, business administration, medical records, management and so on down the list. The medical profession has somewhat vacated this designation, perhaps not wanting a graduate of medicine to suffer a further lack of self esteem.

A doctor of medicine practicing his or her profession was once almost inimitably labeled simply the "doctor", more specifically family doctor, sometimes "doc", or sometimes the surgeon or specialist. Convenient and effective for the purpose of a growing bureaucracy he is later identified as "provider", implying a mediocre, impersonal, commercial identity also described as one of the "health professionals". This is not very gratifying for the degree of involvement and responsibility the physician bears. Only a few can be a medical doctor in view of the requirements of mind and discipline, time, cost and commitment. A new terminology had to develop to accommodate all the non-physicians wanting an identity and a chance to practice medicine, however limited. Hence came the once medical terms now plagiarized for the above reasons; speech pathologist, hearing clinician, physician extender, nurse practitioner, hearing aid consultant, health coordinator, facilitator, paramedic and others.

Burgeoning socialism led by bureaucrats required the culturing of these terminologies to build constituencies; for socialism must offer "promise" to grow; rarely taking responsibility for the effects.



Perhaps on the lighter side, let me address some of the physical identifications of a medical interest. The stethoscope, a device of hearing not understanding, now exists in such profusion that a patient is more likely to have one than the doctor. They are in most rooms and closets. Once an unofficial trapping of the physician, it is now worn in black and grey by serious "health professionals", color coordinated by student nurses and nurse aides. Beepers, communicators, clip boards, name tags and titles all add to the sense of propriety in our environment in medicine today for the non-medical person. White coats, once worn in the lab, or worn by the chief of the service or professor, have lost their identifying character once held by their medical users. One-half length coats were worn by junior residents; full length coats were the province of higher rank. Now days, white coats are worn by the shop foremen in garages, or anyone at the hospital so inclined.

These situations in semantics and visual identity are discussed only to point out that the true identity of the physician relies, not upon titles, garb or gadgets, but is related to the thousands of years and hundreds of generations of heritage and tradition accumulative in knowledge and wisdom in caring for the sick. It is augmented by the strength of character portrayed in the five I's; integrity, intelligence, initiative, intuition, and intensity. These are the qualities of our physicians and are felt by most to be the finest.

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Winston B. Odland, M.D., President
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During the 1980 joint meeting of the American College of Physicians and the South Dakota Society of Internal Medicine, **Theodore H. Sattler, M.D.**, Yankton, was presented the Distinguished Service Award for his many services in the interest of internal medicine and community health. Dr. Sattler has been in private practice at the Yankton Clinic since 1948. He is past chairman of the Dept. of Medicine at Sacred Heart Hospital, professor of Internal Medicine at USD School of Medicine, Chairman of the State Health Coordinating Council, a member of the Board of Directors in the Area Health Education Center Programs and also director of the South Dakota Foundation for Health Care.

* * * *

St. Luke's Hospital of Aberdeen has announced **Chen-fu Chen, M.D.** has joined the medical-dental staff as anesthesiologist. Dr. Chen is originally from Taipei, Taiwan, and received his medical education from Taipei Medical College. He interned at Beekman Downtown Hospital in New York, NY and completed a residency in pathology at Akron City Hospital in Akron, Ohio and a residency in anesthesia at Harlem Hospital Center, in New York. Dr. Chen has moved to Aberdeen from Monticello, NY.

* * * *

Dr. Joel B. Huber, M.D., Redfield, has been named a diplomat of the American Board of Family Practice.

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David A. Smith, M.D., of Yankton, has been named a Fellow of the American of Family Physicians.

* * * *

Hugh Patterson, M.D. has recently began his medical practice at the Lennox Area Medical Center. Dr. Patterson came to Lennox from Roseau, Minnesota. Before practicing in Roseau, he practiced for many years in Slayton, Minnesota. Dr. Patterson received his MD degree at the U. of Minnesota in 1940 and served a one-year internship at the Southern Baptist Hospital in New Orleans. Dr. and Mrs. Patterson have five grown children and eight grandchildren.

* * * *

T. J. Huber, M.D., of Pierre, has been named a diplomate of the American Board of Family Practice.

* * * *

Wm. R. Tschetter, M.D., of Rapid City, has been named a Fellow of the American Academy of Family Physicians.

* * * *

R. V. Avotins, M.D., longtime Faulkton physician, died. Dr. Avotins served the Faulkton community until his retirement in 1975 at the age of 87. Dr. Avotins and his family fled their home in Latvia to Germany in 1944. He worked in Germany as chief surgeon until he and his family immigrated to the United States in 1950. He became staff physician at Redfield State Hospital that year. In 1954, after serving a year's internship and obtaining a South Dakota medical license, he opened his practice in Faulkton. Survivors include: his wife, Alma; one son Peter of Fairfield, Conn.; two daughters, Miriam of Phoenix, Ariz. and Dagmar (Mrs. James) Tibbs of Glen Mills, Penn. and six grandchildren. Also surviving are two daughters from a former marriage; Zeltite Hesse of Bonn, Germany and Mirdsa Kalnins of Lancaster, England.

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tion-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug

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Myxedema Coma—(Hypothyroidism)

It is a true Medical Emergency.
It is rare and is seen in elderly women.

Timothy A. Lamphier, M.D.*

ABSTRACT

Initially the symptoms of Myxedema Coma are quite protean and insidious and it occurs in elderly female patients with hypothyroidism who lapse into coma. This condition is a true medical emergency and fortunately is rare. Early recognition will save lives. Primary myxedema is most frequent in cold winter months.

There are several tests which will distinguish secondary myxedema (pituitary) from primary myxedema (hypothyroidism). Elevated TSH (thyroid stimulating hormone) is elevated in the primary form but not in the secondary or pituitary form.

Secondary myxedema follows destructive lesions of the pituitary gland as is seen in Sheehan's Syndrome or a chromophobe adenoma. Primary myxedema may follow a thyroidectomy, eradication of the thyroid by radioactive iodine, or chronic

thyroiditis.

Of all cases, the primary form makes up the majority of the cases as it amounts to 96 percent of all cases. Since these patients are quite elderly, most complications are cardiac related.

The prodromal symptoms include CO₂ narcosis which is life threatening. Other characteristic symptoms are hypotension, hypothermia, bradycardia, somnolence secondary to hyponatremia, and retention of urine.

In severe myxedema, there is enlargement of the cardiac silhouette. A characteristic chest x-ray is often seen and an EKG may demonstrate marked changes such as low voltage, right-sided heart failure, etc.

Low thyroid levels respond to replacement therapy such as Cytomel or Synthroid.

Carbon dioxide narcosis with attendant alveolar hypotension may account for the high mortality rate.

The mortality in this condition is very high, being 60 to 70 percent, and the etiology for its occurrence is obscure. The symptoms may begin insidiously when an elderly hypothyroid patient lapses into coma. Precipitating causes include tranquilizers, sedatives, narcotics and anesthesia. 96 percent of these

patients have primary (thyroid) myxedema, and only 4 percent have hypothyroidism which is secondary to pituitary insufficiency (secondary myxedema). Primary myxedema or coma is frequent in cold winter months. Pulmonary infections are particularly troublesome because they aggravate an already comprised respiratory system. Congestive heart failure is also frequent.

Several tests are useful in distinguishing pituitary

*Consultant General Surgeon, U.S. Navy, Naval Regional Medical Center, Philadelphia, Pennsylvania.

from thyroid hypothyroidism. One is the determination of thyroid-stimulating hormone (TSH) which is elevated in primary myxedema and falls quickly when thyroid hormone has been replaced. In secondary (pituitary) myxedema, TSH is not elevated in spite of the hypothyroid state.

Thyrotropin releasing hormone (TRH) is elaborated in the hypothalamus and is transported to the pituitary gland via the pituitary portal circulation. It evokes the release of TSH from the pituitary gland into the peripheral circulation which in turn elicits a response from the thyroid gland. The administration of TRH to those with primary myxedema or hypothyroidism results in a greater rise of TSH than in those patients with pituitary deficiency. The metabolic disposal of thyroxine and triiodothyronine is accelerated with infections. The fractional clearance of L-Thyroxine and L-Triiodothyronine from serum is rapid. Binding of thyroid hormone is deficient.

Primary Myxedema may occur after thyroidectomy, eradication of thyroid by radioactive iodine, chronic thyroiditis or the ingestion of goitrogens. The majority of cases are due to atrophy of the gland of unknown causes—possibly due to an autoimmune mechanism.

Secondary Myxedema may follow destructive lesions of the pituitary gland—e.g. postpartum necrosis (Sheehan's Syndrome) or by a chromophobe adenoma. It is manifested by associated disorders of the adrenals and gonads.

The following table summarizes the major differences between Primary and Secondary Myxedema (Coma).

Differentiation of Primary and Secondary Myxedema (Coma)	
Primary (Thyroid) (96% of all cases)	Secondary (Pituitary) (only 4% of cases)
Previous thyroid operation	No previous thyroid operation
Obese	Less obese
Goiter may be present	No goiter present
Hypothermia more common	Hypothermia less common
Increased serum cholesterol	Normal serum cholesterol
Voice coarse	Voice less coarse
Pubic hair present	Pubic hair absent
Sella turcica normal	Sella turcica may be increased in size
Plasma cortisol level normal	Plasma cortisol level decreased
Skin dry and coarse	Skin fine and soft
Heart increased in size	Heart usually small
Normal menses and lactation	Traumatic delivery, no lactation, amenorrhea
No response to TSH*	Good response to TSH*
Good response to Levo-thyroxine without steroids	Poor response to Levothyroxine without steroids
T4 level decreased	T4 level decreased
Serum TSH increased	Serum TSH decreased
*TSH signifies thyroid stimulating hormone	

The Differential Diagnosis: Myxedema can present as unexplained heart failure which fails to respond to digitalis or diuretics, "idiopathic" hyperlipemia and unexplained ascites. Myxedematous changes in the tongue may be confused with the thick tongue of primary amyloidosis. Profound myxedema may present the clinical picture of primary psychosis or brain tumor.

Complications: As these patients are usually elderly females, complications are usually cardiac-related. These patients are prone to develop infection. "Myxedema Madness" may actually be organic psychosis. It is well-known that these patients are extremely sensitive to opiate medication. When one encounters a refractory hyponatremia the causative factor may be a defect in the distal tubular reabsorption of sodium and water by the kidney or to inappropriate secretion of antidiuretic hormone. It must be remembered that these patients metabolize drugs slowly.

The prodromal symptoms include CO₂ narcosis, retention of urine, somnolence secondary to hyponatremia bradycardia, hypotension and hypothermia. This hypotension responds poorly to pressor agents.

Often the history and physical examination will almost pinpoint the diagnosis, and it should include:

- Previous thyroid surgery
- Previous use of radioactive iodine
- Exophthalmus
- Hypoventilation secondary to depression of respiratory center, decreased cerebral flow, decreased O₂, decreased glucose consumption
- Pretibial edema
- Puffy face
- Hoarse voice
- Profound stupor
- Dry, cold, hyperkeratotic skin
- Scaly elbows and knees
- Obesity
- Alopecia
- Bradycardia
- Yellow skin
- Peri-orbital edema
- Pasty complexion
- Sparsity of lateral portions of eyebrows
- Obtundity-confusion-lethargy-disorientation-somnolence-twitching—??Cheyne Stokes
- Nasal stuffiness
- Conduction hearing loss (deafness)
- Enlarged tongue
- Hypoxia
- Hypercapnia
- Shallow rapid respiration

- z. Cachexia
- aa. Circumoral and nailbed cyanosis
- bb. Hypoglycemia secondary to pituitary deficiency
- cc. Increased appetite
- dd. Inelastic chest wall resulting in decreased lung compliance and respiratory mechanics
- ee. Decreased taste and smell
- ff. Amenorrhea
- gg. Slow speech
- hh. Constipation
- ii. Anginal pain
- jj. Thinning of hair
- kk. Thin brittle nails
- ll. Lethargy
- mm. Delayed return of deep tendon reflexes
- nn. Absence of sweating
- oo. Effusions into pleura-peritoneum-pericardium and joints
- pp. Aches and pains

Hyponatremia is the result of inappropriate secretion of antidiuretic hormone.

Enlargement of the cardiac silhouette on the chest roentgenogram, severe bradycardia, and hypotension (which is poorly responsive to pressor amines) may be seen in severe myxedema.

Listed below is the necessary laboratory data:

LABORATORY:

- 1. CBC—Hct—Sedimentation Rate
- 2. Urinalysis
- 3. BCP₁₆ (Blood chemical profile ₁₆ = SMA₁₂ plus electrolyte profile)
- 4. T₃ (Resin Uptake)
- 5. T₄ (RIA)
- 6. Creatinine
- 7. TSH by radio-immuno assay
- 8. L-Thyroxine Blood Level ↓
- 9. L-Triiodothyronine Blood Level ↓
- 10. Free Thyroxine—(normal 1.0 to 2.1) ↓
- 11. Lipid Blood Level
- 12. Cholesterol Blood Level
- 13. Protein Spinal Fluid
- 14. EKG—
 - Low Voltage
 - Bradycardia
 - Prolonged QT and P.R. Intervals
 - Diffuse T wave depression
 - Right Ventricular Hypertrophy
 - Right sided Heart Failure
- 15. Chest x-ray—
 - Pleural effusion
 - Pericardial effusion
 - Enlargement of cardiac silhouette
- 16. CO₂ (elevated) ↑
- 17. Depressed pO₂ and pH ↓

Elevated

- 18. Blood Sodium level (Hyponatremia)—water restriction
- 19. CPK (elevated) ↑
- 20. SGOT—LDH—Aldolase—(elevated) ↑
- 21. Chest x-ray—enlarged cardiac silhouette
- 22. Abdominal flat plate (x-ray)
 - ??Gaseous Distention with bloating and constipation
- 23. Culture and Sensitivities of:
 - a. Tracheal aspirates
 - b. CSF—(cerebrospinal fluid)
 - c. Blood
 - d. Urine
- 24. EEG
- 25. Cerebro-spinal fluid:—
 - a. Pressure increased
 - b. Increased protein concentration
- 26. Hypoglycemia (daily check)
- 27. Arterial blood gases—daily
- 28. Sella turcica x-rays for secondary myxedema

In treatment, the management of complications is as important as the thyroid replacement itself.

A suggested protocol of treatment is as follows:

- 1. Automatic mechanical ventilatory assistance (?? cuffed endotracheal tube)
- 2. ?? Tracheostomy
- 3. Check temperature with a **special low recording thermometer**
- 4. Steroids for secondary hypothyroidism secondary myxedema and adrenal insufficiency due to deprivation of ACTH (Hydrocortisone 100 to 200 mg. daily)
- 5. **Replacement Therapy:**
 - (a) Cytomel (L-Triidothronine) 10 mg. every 8 hours I.V. (rapid return to euthyroid (eumetabolic state).
 - (i) If given with pressor amines order only 12.5 mg. every 12 hours to prevent arrhythmias
 - OR
 - (b) Synthroid (L-Thyroxine)—0.5 mg. I.V. followed by 0.05 mg. I.V. or 0.1 mg. orally each day thereafter.
 - (i) One dose usually suffices for the first 5 to 7 days
 - (ii) Clinical improvement should be expected in 6 to 12 hours
 - OR
 - (c) Levothyroxine I.V. 400 to 500 mg. I.V. combined with hydrocortisone 300 I.V. and then taper off quickly
- 6. A 3-way Foley catheter for urinary retention (use Neosporin GU irrigant to avoid infection)
- 7. C.V.P. Monitoring
- 8. ??? Hypertonic saline for salt deprivation
- 9. If infection, order broad spectrum antibiotics

10. **Avoid sedatives and narcotics**
11. **Constant cardiac monitoring**
12. CSF— { pressure is increased.
increased protein concentration
13. Daily check for **hypoglycemia** (order intravenous glucose for hypoglycemia).
14. With hypothyroidism of pituitary origin, give hydrocortisone 100 to 200 mg. daily.
15. Active warming is contraindicated as it may precipitate peripheral vascular collapse.
16. After emergency has been under control, order oral replacement thyroid hormone—e.g. desiccated thyroid 1 Gm. per day or L-Thyroxine 0.1 mg. per day increasing to 0.15 mg. to 0.3 mg. of L-Thyroxine.

SUMMARY

1. Myxedema coma is a true medical emergency and it demands immediate treatment as outlined above in the suggested protocol.
2. The difference between primary and secondary myxedema have been pointed out.
3. The complications of the coma are just as important and demand immediate care—as well as the thyroid replacement itself.
4. Alveolar hypoventilation with carbon dioxide narcosis may account for the high mortality rate.
5. There is outlined below a detailed flow sheet covering signs, symptoms, important laboratory data and treatment for easy monitoring.

Flow Sheet Myxedema Coma Early Signs and Symptoms

Hypothermia
Weakness
Fatigue
Dry-Cold Hyperkeratotic Skin
Scaly Elbows & Knees
Alopecia
Nervousness
Thinning Hair
Thin Brittle Nails
Lethargy
Delayed Deep Tendon Reflexes
Pallor
Poor Turgor of Mucosa
Cold Intolerance
Headache
Retention of Urine
CO₂ Narcosis
Menorrhagia

Flow Sheet Myxedema Coma Late Signs And Symptoms Physical Examination

Obtundity
Confusion
Hypotension—Poorly responsive to pressor amines
Disorientation
Somnolence
Twitching
?? Cheyne-Stokes

Flow Sheet Myxedema Coma Late Signs And Symptoms Physical Examination

Slow Speech—Voice Coarse
Hard Pitting Pretibial Edema
Profound Stupor
Bradycardia—Severe
Yellow Skin
Peri-orbital Edema
Pasty Complexion
Hypothermia (80% of cases)
Effusions into Pleura—Peritoneum-Pericardium-Joints
Puffy Face
Amenorrhea
Aches and Pains
Decreased Taste & Smell
Sparsity of Lateral Portions of Eyebrows
Nasal Stuffiness
Conduction Hearing Loss—Deafness
Enlarged Tongue
Shallow Rapid Respiration
Cachexia
Circumoral and Nailbed Cyanosis
Increased Appetite
Constipation
Anginal Pain
Exophthalmus
Hypoventilation
Inelastic Chest Wall Decreased Lung Compliance—Decreased Respiratory Mechanics

Flow Sheet Myxedema Coma Laboratory

Date
Time
T₃ ↓

T_4 ↓
 Radioactive Uptake ↓
 TSH
 a. High in Primary Form
 b. Low in Secondary Form
 Free Thyroxine
 17 Ketosteroids
 CBC—Hct.—Hg.
 Macrocytic Anemia
 a. MCV
 b. MCH
 c. MCHC
 Sed Rate
 Urinalysis
 BCP₁₆
 CPK ↑
 CO₂ ↑
 Creatinine
 Blood NA Level ↓
 Carotene Blood Level ↑

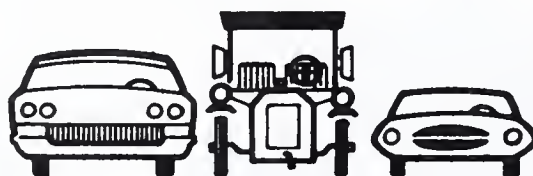
Flow Sheet Myxedema Coma Laboratory

Lipid Blood Level ↑
 Plasma Cholesterol ↑
 Protein in CSF ↑
 CSF Pressure ↑
 EKG Monitor
 a. ?? Congestive Failure
 Chest X-ray
 Cardiac Silhouette
 (Water Bottle Heart)
 Arterial Gases Daily
 a. PO₂ ↓ —Hypoxia
 b. pH ↓
 c. PCO₂ ↑ —Hypercapnia
 Flat Plate Abdomen
 a. Gaseous Distention
 b. Constipation
 Culture and Sensitivity
 a. Tracheal Aspirates
 b. CSF
 c. Blood
 d. Urine
 EEG
 SGOT
 LDH
 Aldolase

Flow Sheet Myxedema Coma Treatment

Date
 Time

Cytomel 10 mg IV q 8 h
 Hydrocortisone 100-200 mg daily
 Avoid Sedatives & Narcotics
 Hypoglycemia (I.V. Glucose)
 Core Temperature—Special Thermometer
 3-way Foley Catheter
 Intake
 Output
 CVP Monitor
 Antibiotics if Infection
 Mechanical Ventilation Assistance Endotra-
 cheal
 Tube
 ?? Tracheostomy
 Daily check for hypoglycemia ?? IV Glucose
 Secondary Myxedema-Steroids
 No Active Warming
 Hypertonic Saline for Salt Deprivation
 Constant Cardiac Monitoring
 Monitor Blood Gases
 Monitor by Suppression Serum TSH
 Hypotension—Poorly responsive to Pressor
 Amines
 Never interrupt Therapy Because Relapses Oc-
 cur
 Treatment is Indefinite



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Newborn With Enlarged Head And Abnormal Computer Tomographic Scan Of Cranium

C. K. Hansen, M.D.*

K. A. Kelts, M.D.**
Discussers

J. F. Barlow, M.D.***
Editor

Case No. 867 579

This premature 32-week gestational age, 2098 gm Caucasian male was transported from another hospital to the intensive nursery at Sioux Valley Hospital with a diagnosis of hydrocephalus.

The mother was a 29-year-old gravida II para I. The mother had had pneumonia a month prior to admission. She was treated for morning sickness with benedectin and for premature labor by terbutaline. It was known by sonogram that the fetus did have head enlargement (macrocephaly) before delivery. After the mother went into labor, there was a decrease in fetal heart tones and a Caesarean section was performed. The infant had Apgar scores of 2 at 1 minute and 3 at 5 minutes. The baby was thought to have massive congenital hydrocephalus. The head circumference was 41.5 cm at birth and the baby became pink on the ventilator.

PHYSICAL EXAMINATION: The infant was lethargic with little spontaneous movement and on a ventilator. There was a markedly enlarged head with a circumference 41.5 cm. There was transillumination of the head in spotty areas with a large area of transillumination in the right parietal and occipital areas. The left eye was proptotic. Both pupils were dilated and the corneas were somewhat hazy. There was a pale orange disc seen but no vessels and no chorioretinitis could definitely be seen. The baby showed little movement in all extremities.

There were no heart murmurs and the peripheral pulses were easily palpable and equal. The liver was enlarged to 4 cm below the right costal margin and extended across the midline. The spleen was palpable 0.5 cm below the left costal margin. Both kidneys were palpable. There were no abdominal masses. There were three umbilical cord blood vessels noted. The infant was a normal male with a left testis descended. The hips were unremarkable. Neurological examination by a consultant revealed marked hypotonia and flaccidity in addition to marked macrocephaly. All the fontanelles were enlarged and the sutures were spread. The eyes were called divergent and mildly proptotic. No abnormality of the Moro or other reflexes were noted. There was mild withdrawal of all four extremities. He was pink but lethargic. There was generalized edema. There were spotty areas of transillumination anteriorly, occipitally and in the right parietal area.

LABORATORY DATA: Urinalysis: yellow, clear; specific gravity 1.015; pH 5.0; negative for glucose, ketone bodies, bile, hemoglobin, and glucose; sediment, occasional granular casts. Hemoglobin 10.3 gm/dl; red count 2.84 million/mm³; hematocrit 33 vol/dl; mean corpuscular hemoglobin 36 micromicrograms; mean corpuscular volume 113 cubic micra; mean corpuscular hemoglobin concentration 32%; total leukocyte count 11,000/mm³, (corrected for nucleated red cells) with a differential of 16% segmented neutrophils; 14% neutrophilic bands; 3% eosinophils; and 67% mature lymphocytes. There were 175 nucleated red cells per 100 white cells. The red cells showed marked anisocytosis and moderate polychromatophilia. Platelet count was 210,000/mm³. Arterial blood gases: pH 7.25; PCO₂ 33 torr; CO₂ content 15 mm/L; PO₂ 38 torr, O₂ saturation 63% at oxygen of 96%, base deficit 11 meq/L. Serum calcium 7.3 mg/dl (normal 9.0-11.5 mg/dl). Sodium 132 meq/L. Potassium 5.3 meq/L. Blood type - RH₀ (D) positive. A direct antiglobulin test was negative. The mother was O RH₀ (D) Positive. A screen of the maternal serum for irregular or

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atypical antibodies was negative. A computer tomographic scan (CT) of the brain showed diffuse brain calcification and enlargement (Fig. 1). Calcifications were present on the CT scan as well as skull films. A chest film showed minimal newborn atelectasis. Chromosome studies revealed a normal karyotype in the blood. Cultures of urine for virus were negative. The patient died on the second hospital day.

DR. HANSEN: The marked enlargement of the head in this preterm male infant makes the differential diagnosis essentially one of hydrocephalus. In order to understand the classification of hydrocephalus, one must consider normal spinal fluid physiology. Cerebrospinal fluid production depends upon active transport of ions such as sodium across the choroid plexus of the ventricles into the ventricular cavities. The net flow of fluid is from the lateral ventricles through the foramina of Monro into the third ventricle and then through the aqueduct of Sylvius into the fourth ventricle. The cerebrospinal fluid gains access to the subarachnoid space through the lateral foramina of Luschka and the medial foramen of Magendie. The cerebrospinal fluid courses through the subarachnoid space over the convexities of the cerebral hemispheres and is absorbed into the venous circulation of the sinuses of the brain and partially over the spinal cord. There is apparently some absorption through the ependymal lining of the ventricle.

Hydrocephalus is usually due to obstruction of flow of cerebrospinal fluid either between the ventricles of the central nervous system or within the subarachnoid space. Another mechanism of hydrocephalus is ineffective absorption of cerebrospinal fluid from the subarachnoid villi into the venous circulation. Very rarely hydrocephalus may be caused by overproduction of cerebrospinal fluid by a papilloma of the choroid plexus that is actively secreting spinal fluid.

Hydrocephalus can be classified anatomically as obstructive or non-communicating hydrocephalus or as communicating hydrocephalus. In obstructive hydrocephalus there is a block of flow of cerebrospinal fluid within the ventricular system so that the progress of the fluid flow into the subarachnoid spaces is impeded. There is dilatation of the ventricular system proximal to the block. In communicating hydrocephalus the block of cerebrospinal fluid is either in the subarachnoid space itself, commonly around the brain stem, or over the convexities of the brain.

Congenital aqueductal stenosis causes obstructive hydrocephalus of non-communicating type. In this condition the aqueduct of Sylvius is narrowed, atretic, or replaced by multiple channels called forks that end blindly. This condition may be inherited as an x-linked recessive trait or be the result of inflammation in the periaqueductal region. Mumps,

influenza and parainfluenza viruses have been thought to be responsible for this type of inflammation.

Rarely an extrinsic lesion, posterior to the brain stem, can produce compression producing obstructive hydrocephalus. One such anomaly is a congenital aneurysm of the great vein of Galen. This venous malformation may be associated with heart failure and a bruit over the vertex of the skull.

Traumatic rupture of bridging veins over the surface of the cerebellum communicating with the transverse sinus may produce a posterior subdural hematoma which can also produce compression of the aqueduct and obstructive hydrocephalus. This type of trauma usually results from birth injury.

The Dandy-Walker malformation is a congenital defect in the midline cerebellar structures including atresia of the foramina of Luschka and Magendie producing obstructive hydrocephalus due to a large, cystic dilatation of the fourth ventricle. Occipital translumination is a feature of this Dandy-Walker abnormality.

Midline infratentorial brain tumors may cause compression of the aqueduct producing obstruction to cerebrospinal fluid flow and hydrocephalus. Tumors such as medulloblastoma, teratomas, ependymomas and astrocytomas of the cerebellum and craniopharyngiomas most commonly occur postnatally and can produce acquired non-communicating obstructive hydrocephalus.

The Arnold-Chiari malformation obstructs the subarachnoid fluid pathways around the brain stem by downward displacement of the cerebellum and the brain stem through the foramen magnum producing a communicating hydrocephalus. This malformation is sometimes associated with a lumbar meningomyelocele and is of unknown etiology.

Fibrosis and adhesions resulting in obstruction of the subarachnoid pathways can produce communicating hydrocephalus. Such adhesions may result from subarachnoid hemorrhage or post inflammatory states such as bacterial meningitis, toxoplasmosis or cytomegalovirus.

The differential diagnosis of hydrocephalus just described includes both congenital and acquired causes. The infant presented today was known to have hydrocephalus prior to birth and thus has a congenital hydrocephalus. Congenital anomalies or intrauterine infection appear to be much more likely causes for the development of hydrocephalus than the acquired forms such as subarachnoid hemorrhage or subdural hematoma associated with birth trauma in this case. The maternal drug history does not suggest a cause for the hydrocephalus. Bendectin is a commonly used anti-emetic and is not associated with hydrocephalus. It is not known what drugs



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DESCRIPTION

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This product is prepared from human venous plasma. Each individual unit of plasma has been found nonreactive for hepatitis B surface antigen using the radioimmunoassay method of counter-electrophoresis.

INDICATIONS

Treatment of rabies, once clinical disease becomes apparent, is rarely if ever successful. Rabies vaccine (duck-embryo origin, Lilly Laboratories) with or without Rabies Immune Globulin (Human)—Hyperab[®] should, therefore, be given to all persons suspected of exposure to rabies, particularly to severe exposure. Whenever possible, Hyperab[®] globulin should be injected as promptly as possible after exposure. If initiation of treatment is delayed for any reason, however, Rabies Immune Globulin (Human) should be given just the same, regardless of the interval between exposure and treatment.

Rabies virus is usually transmitted by the bite of a rabid animal, but can occasionally penetrate abraded skin with the saliva of infected animals. Progress of the virus after exposure is believed to follow a neural pathway, and the time between exposure and clinical rabies is a function of the proximity of the bite (or abrasion) to the central nervous system and the dose of virus injected. The incubation is usually 2 to 6 weeks, but can be longer. After severe bites about the head and neck, it may be as short as 10 days.

After initiation of the vaccine series, it takes 2 weeks or longer for development of immunity to rabies. Since most vaccine failures have occurred in cases of severe exposure, the value of immediate immunization with preformed rabies antibody cannot be over-emphasized.

Recommendations for use of passive and/or active immunization after exposure to an animal suspected of having rabies were detailed by WHO, and by the US Public Health Service Advisory Committee on Immunization Practices (ACIP).

INJECTION PROCEDURE

A portion of the Hyperab[®] globulin dose should be used to infiltrate the wound. The rest is injected intramuscularly.

CONTRAINDICATIONS

Rabies Immune Globulin (Human)—Hyperab[®] is contraindicated in repeated doses, once vaccine treatment has been initiated. Repeating the dose may bring about interference with full expression of active immunity expected from the vaccine. Hyperab[®] globulin is also contraindicated in individuals who are known to have an allergic response to gamma globulin or thimerosal.

PRECAUTIONS

NEVER ADMINISTER Hyperab[®] globulin INTRAVENOUSLY.

ADVERSE REACTIONS

Slight soreness at the site of injection, and slight temperature elevation, may be noted at times. Sensitization to repeated injections of human globulin is extremely rare.

In the course of routine injections of a large number of persons with human gamma globulin, there have been a few isolated occurrences of angioneurotic edema, nephrotic syndrome, and anaphylactic shock after injection. Because of their rarity, it is difficult to determine whether such reactions are incidental, or causally related to the gamma globulin.

No instances of transmission of hepatitis B (homologous serum jaundice) have been reported from the use of human gamma globulin prepared by the fractionation methods employed by Cutter Laboratories, Inc.

HOW SUPPLIED

Rabies Immune Globulin (Human)—Hyperab[®] is packaged in 2-ml. and 10-ml. vials with a potency of 150 International Units per ml. (IU/ml.). The 2-ml. vial contains a total of 300 IU which is sufficient for a child weighing 15 kg (33 lb.). The 10-ml. vial contains a total of 1500 IU which is sufficient for an adult weighing 75 kg (165 lb.).

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the mother received for her pneumonia prenatally. Terbutaline was not used until the end of her gestation.

The infant was apparently not appropriate for the gestational age in weight but because of decreased fetal heart tones, a Caesarian section was performed and the infant had Apgar scores of 2 at one minute and 3 at five minutes. Arterial blood gases showed marked hypoxia in spite of high FiO_2 concentrations on a ventilator despite a normal cardiac examination, the presence of peripheral pulses, and only minimal atelectasis on chest x-ray. The peripheral blood showed an anemia with nucleated red cells. There was hepatosplenomegaly on physical examination. There was no evidence of icterus or laboratory evidence of blood group incompatibility.

Of special interest is the spotty transillumination of the skull noted in the protocol, and the brain calcifications noted both on skull films and CT scan of the brain. The combination of a congenital hydrocephalus with diffuse brain calcification limits the diagnostic possibilities. Brain calcification is seen commonly in two congenital infections—cytomegalovirus infection and congenital toxoplasmosis.

In congenital cytomegalovirus infection, microcephaly and psychomotor retardation usually occur. Hydrocephalus is rare in this infection but does occur. Cerebral calcifications are classically periventricular in location. Hepatosplenomegaly and icterus are very common in the neonatal period; and, when there is no evidence of hepatitis, are probably due to extramedullary hematopoiesis. Thrombocytopenia and anemia are characteristic of cytomegalovirus infection. The infant in our case meets only some of these criteria for this disease and one must remember that hydrocephalus is rare in congenital cytomegalovirus infection. The CT scan of the brain also reveals diffuse calcifications rather than periventricular calcifications.

Another congenital infection, caused by a protozoan named *Toxoplasma gondii*, produces a zoonosis which affects many members of the animal kingdom but only the cat is a complete host for this organism. Other vertebrates are commonly infected but do not transmit the disease as does the cat. The domestic cat is the most common source shedding infective oocysts in the feces. Congenital toxoplasmosis is acquired transplacentally. Only 70% of infants born with the infestation will manifest the clinical illness in the neonatal period. Infected infants fall into two major groups. Infants in the first group have generalized disease which is manifested mainly as a low birth weight, hepatosplenomegaly, icterus, and anemia. Infants in the second group have primarily neurologic disease which is manifested mainly as abnormal spinal fluid, convulsions,

intracranial calcifications, and hydrocephalus. Chorioretinitis is the most common lesion in congenital toxoplasmosis occurring in up to 94% of the infections. This is seen in both of the major groups that I have described, but is not apparent in the neonatal period. The brain calcifications of congenital toxoplasmosis are characteristically described as diffuse intracerebral calcifications in contrast to periventricular calcifications seen in cytomegalovirus infection. This infant did present with a diffuse brain calcification in the absence of chorioretinitis. The absence of the latter does not preclude congenital toxoplasmosis in spite of the fact that it is a frequent finding. The infant also seems to have clinical findings which are present in both of the major groups of toxoplasmosis which I have described. For example, hepatosplenomegaly and anemia occur in the generalized disease group and intracranial calcification and hydrocephalus in the neurologic disease group.

Careful examination of this infant's CT scan of the brain might suggest a mass lesion with calcification as the midline does appear to be shifted and the large symmetric fluid filled ventricles which one might expect in hydrocephalus are not seen. (Fig. 1) Congenital neoplasms may account for mass effect and brain calcification. Ependymomas, craniopharyngiomas, and teratomas all are tumors which



Figure 1
CT scan of brain revealing diffuse calcification and irregular cystic areas.

could produce calcification. An ependymoma arising in the 4th ventricle may be especially suspect in that it commonly is associated with calcification. However, supratentorial tumors are unlikely to be found congenitally since these are seen usually as acquired tumors later in childhood.

Dr. Hansen's Diagnoses Congenital Toxoplasmosis

DR. KELTS: I would like to approach this discussion from the viewpoint of the causes of macrocephaly, enlargement of the head. The following is a list of such abnormalities:

- I. Hydrocephalus/Hydranencephaly
 - A. Developmental Disorders—rostral neural tube malformation, e.g., Arnold-Chiari malformation, aqueductal stenosis, and hydranencephalus
 - B. Mass Lesions—neoplasm, arteriovenous malformations, cysts
 - C. Intrauterine Infections of the TORCHS Group (Toxoplasmosis, Other, Rubella, Cytomegalovirus, Herpes, Syphilis)
- II. Megalencephaly
 - A. Primary—syndrome of mental retardation, seizures, and mild neurologic defects
 - B. Cerebral Gigantism (Soto's syndrome)
 - C. Achondroplasia
 - D. Neurocutaneous syndromes
- III. Normal Variant

The description of hydrocephalus and the dynamics of the ventricular system have been well discussed. The formation of cerebrospinal fluid is at a rate of 0.3 to 0.4 ml/minute. The total cerebrospinal fluid of the newborn is 50 ml and of an adult 150 mls. It should be noted that the arterial pulsations of the choroid plexus act as a pump. Drainage of the spinal fluid into the venous sinuses by the arachnoid villi is by a one-way valve mechanism. As has been pointed out, hydrocephalus is divided into communicating and noncommunicating types.

In the Arnold-Chiari malformation, 40% of the children have hydrocephalus. Due to an abnormal pontine flexure, the medulla is displaced caudally into the spinal canal and is often accompanied by an extension of cerebellar tissue through the foramen magnum. Other abnormalities include beaked colliculi, lumbosacral myelomeningocele, and an occasional encephalocele.

Aqueductal stenosis causes 20% of cases of noncommunicating hydrocephalus. This may result from congenital stenosis, atresia or forking, or periaqueductal gliosis from inflammation. The method by which intrauterine infections produce gliosis is only partially understood. The microorganisms in this group (TORCHS) including toxoplasmosis, other viruses, rubella, cytomegalovirus, herpes and

syphilis, must attack the infant during the blastula stage to produce damage in the periaqueductal tissue either by direct cytotoxicity or vascular lesions producing cellular necrosis and calcification. It is important to realize that the time of the insult of the agent is extremely important in regard to the effects that are produced in the infant. In fact, the timing may be even more important in predicting the outcome of the abnormality than the actual agent producing the disease process.

Different causes of megalencephaly include cerebral gigantism, achondroplasia, neurocutaneous syndrome, and primary megalencephaly. In cerebral gigantism, the baby is diffusely large and has an enlarged head from birth. Other dysmorphic features include an unusual facies, frontal bossing, widely spaced eyes, and a large prominent jaw. Developmental delays and mental retardation frequently are present. In achondroplasia, the mild hydrocephalus is associated with a malformed skull. Neurocutaneous syndromes, such as neurofibromatosis, may produce a large brain due to excessive proliferation of some of the elements of the gray and white matter, often associated with ectopias due to abnormal migration of tissue. Other markers of neurofibromatosis are cafe-au-lait spots, cutaneous neurofibromas, and a positive family history. The primary megalencephaly syndrome includes mental retardation, seizures, and cortico-spinal and cerebellar signs in addition to a large head. In this disease, there is a large anterior fontanel but no frontal bossing or signs of increased intracranial pressure.

Careful examination of the CT scan reveals markedly abnormal organization of intracranial tissue with poor differentiation of the two hemispheres, the absence of definite ventricles, unrecognizable CNS structures, cysts, and areas of calcification. Therefore, the scan fails to demonstrate hydrocephalus and does demonstrate markedly abnormal cerebral architecture and calcification, thus providing presumptive evidence for an intrauterine infection during the early stages of embryogenesis.

Dr. Kelts' Diagnosis Early Intrauterine Infection (TORCHS)

DR. BARLOW: On entering the skull, the dura and meninges were adherent to the brain which was also adherent to the skull. On the left side, tissue connected with the pharynx had to be severed in order to remove the cerebral hemispheres. The fresh brain weighed 700 gm. and the cerebellum and brain stem appeared normal. Cut section of the brain revealed the principle abnormality in this case (Fig. 2). The cerebral hemispheres were replaced by neoplasm with gritty calcification and a few small irregular cysts. The hemispheres were asymmetrical, but any

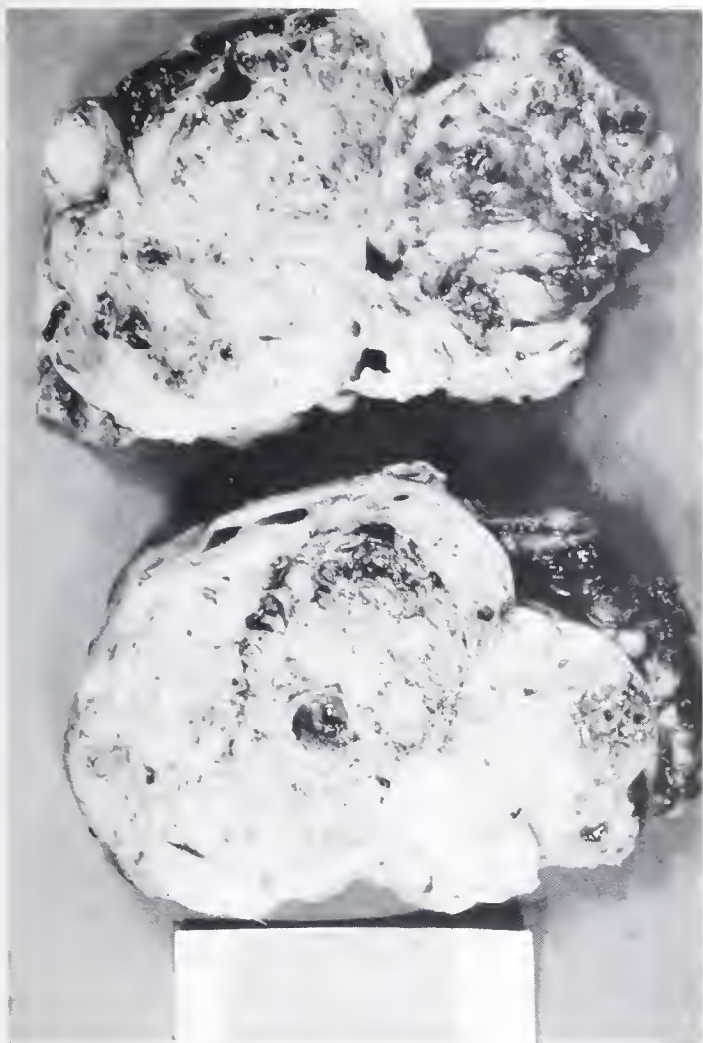


Figure 2

Cross section of cerebral hemispheres demonstrating complete replacement by neoplasm.

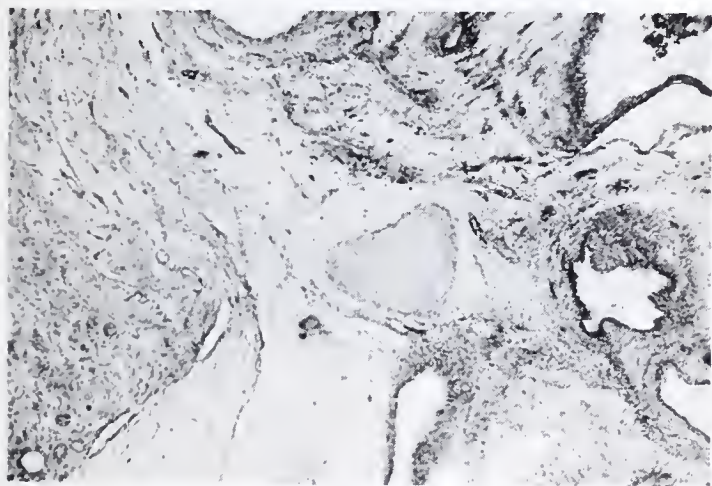


Figure 3

Histologic section of teratoma revealing fragment of cartilage in midst of mature glia and surrounded by cysts lined by various types of epithelium.

residual architecture of normal cerebrum was not noted. On microscopic examination the tumor proved to be an immature teratoma. Multiple different types of tissue from embryonal mesenchyme to adult neural tissue or mature cartilage were seen. Elements of endoderm, mesoderm, and ectoderm were represented when multiple sections were taken (Fig. 3). In addition, the patient did have pulmonary

atelectasis, focal early bronchopneumonia and some focal necrosis of the mucosa and submucosa of the colon probably due to shock and hypoxia. There were bilateral undescended testes.

Teratomas are neoplasms composed of multiple tissues. The tissues may be mature or immature. In general, the greater the amount of immature tissue, the more malignant the potential of the lesion. In most organs, mature teratomas have a better prognosis than those with immature tissues, although this is not always the case. All three germ layers can usually be found in teratomas, but representatives of all three may be difficult to find in some cases. In this case, the three germ layers can be noted in multiple sections. It is important to note that other types of germ cell tumors, such as endodermal sinus tumor, germinomas (dysgerminoma, seminoma), embryonal carcinoma, and choriocarcinoma may occur along with the mature or immature teratomatous elements. None of these were seen in this case.

Teratomas most commonly arise in the gonads but they have been reported in almost any organ in the body with a predilection for the midline structures of the pharynx, base of the brain, pineal region, mediastinum, thyroid, heart and its coverings, retroperitoneum and sacrococcygeal region in infants.

In the brain, teratomas are often classified in a group of congenital tumors of maldevelopmental origin. They originate in the midline in the sprapharyngeal or pineal region. Such tumors include the teratomas, dermoid cysts, epidermoid cysts, choles- teatomas, and craniopharyngiomas (adamantinoma, ameloblastoma). The teratomas arising in this region may extend into the buccal or pharyngeal cavity and are often called teratoma epignathi. Very rarely they may extend into the cranial cavity and replace the entire brain as in this case.

Teratomas of the brain constitute only 0.5% of total brain tumors, but compose 2% of brain tumors under the age of 15. Males usually outnumber females except in the neonatal age group.

Sweet reviewed 156 teratomatous tumors in 1940 but only 12 were under one year of age. Tamura reviewed 32 cases from the literature and added 3 from stillborn fetuses. Dystocia during labor and rupture of the skull were complications. Eight of the 35 were huge completely or almost completely replacing the cerebral hemispheres.

I believe this case represents a teratoma arising in the pharyngeal region with massive extension into and replacement of the cerebral hemispheres.

FINAL ANATOMIC DIAGNOSIS IMMATURE TERATOMA, COMPLETELY REPLACING THE CEREBRAL HEMISPHERES

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**S
D**

Letters To The Editor

I believe the article in the January Journal entitled, "Increase In Physicians Affects Practice Arrangements", was worthwhile.

South Dakota has made dramatic progress in the more sophisticated types of medical procedures, partly due to the Medical school and superb faculty, but I feel we must watch the economics of the whole situation.

We are, going to have more MD's, and I hope some will locate in the rural areas; but what kind of facilities will they need to practice the type of medicine they have been exposed to and who will pay the bill?

Roscoe E. Dean, M.D.
Wessington Springs, S.D.

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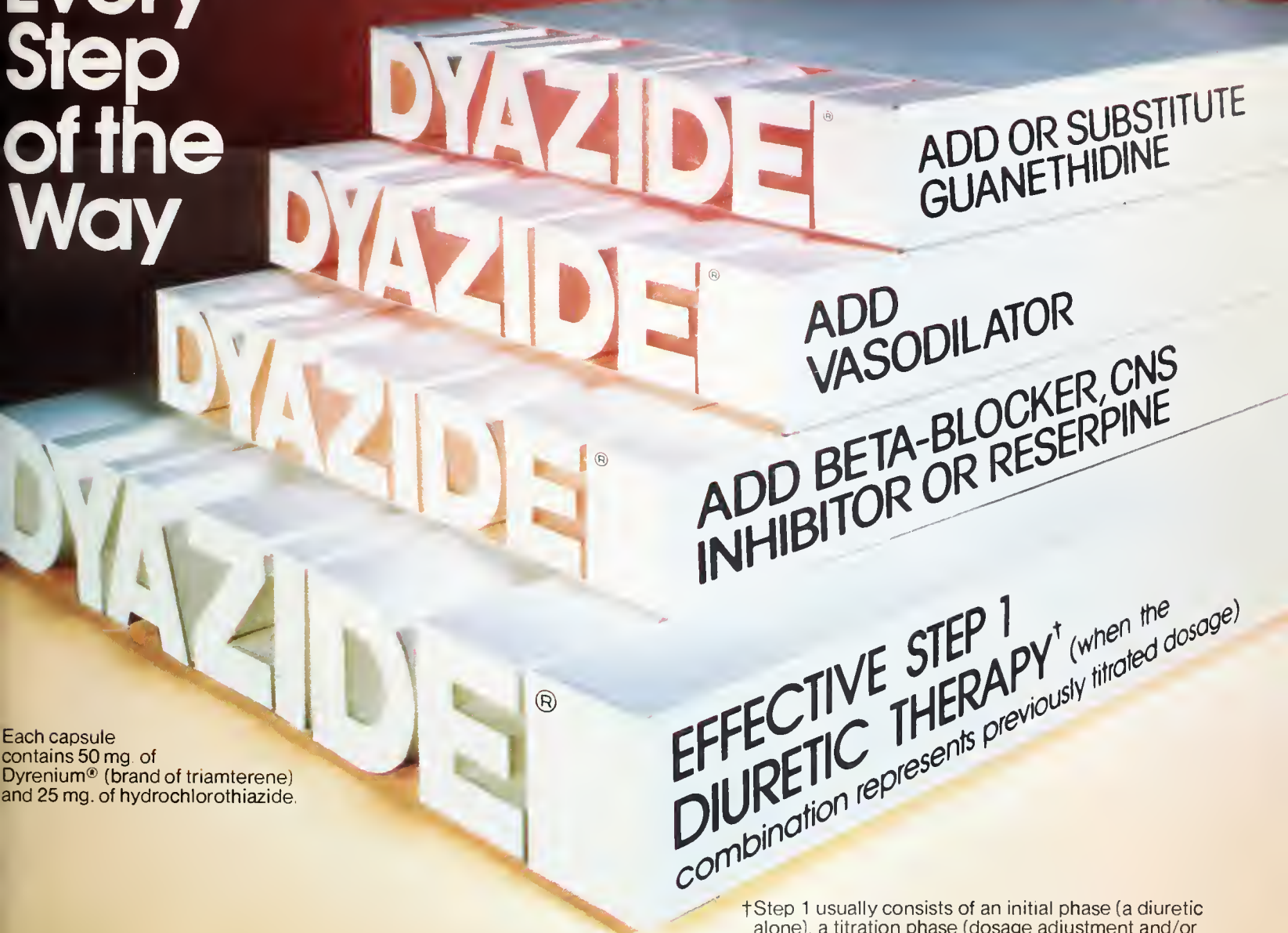
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[†]Step 1 usually consists of an initial phase (a diuretic alone), a titration phase (dosage adjustment and/or addition of a K⁺ supplement or K⁺-sparing agent) and a maintenance phase (a diuretic alone or in combination with a K⁺ supplement or K⁺-sparing agent).

Serum K⁺ and BUN should be checked periodically (see Warnings).

Before prescribing, see complete prescribing information in SK&F Co. literature or PDR. A brief summary follows:

WARNING

This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K⁺ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K⁺ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, throm-

bocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K⁺ frequently, both can cause K⁺ retention and elevated serum K⁺. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Anti-hypertensive effect may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with

possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia, although uncommon, has been reported. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function.

Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth, anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components.

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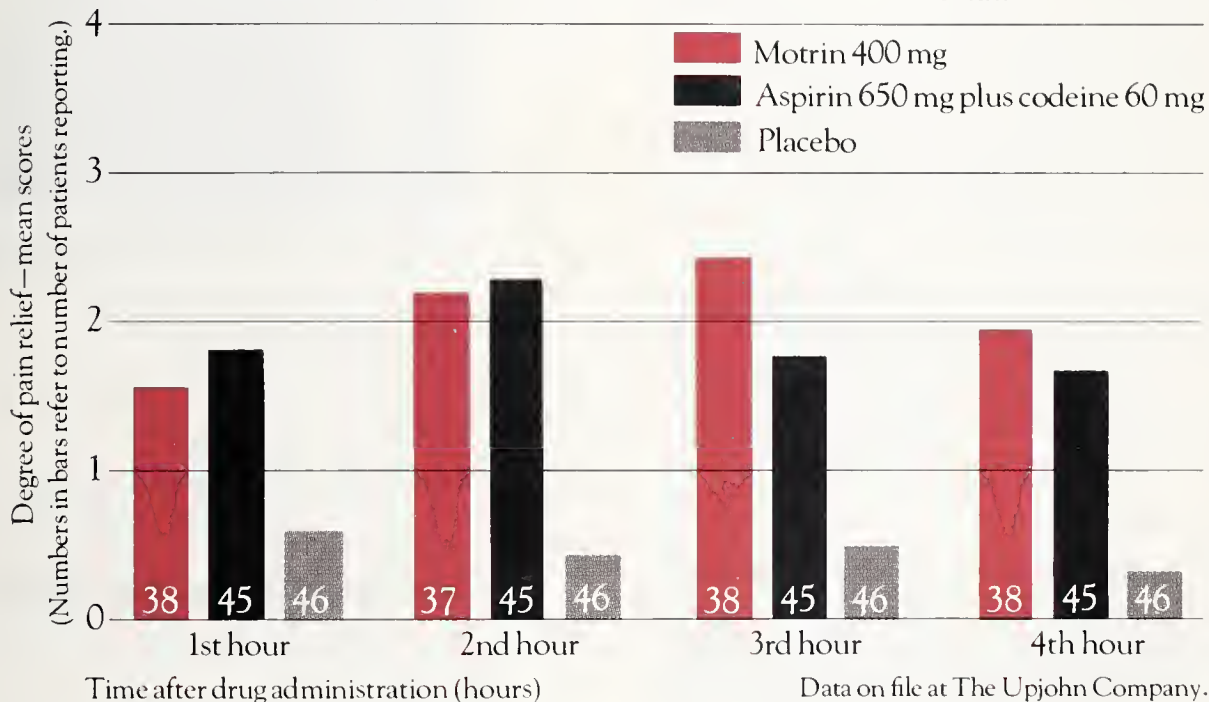
In this double-blind, placebo-controlled, randomized study, no statistically significant difference in relief of pain was noted at 1, 2, and 4 hours between the Motrin and aspirin-with-codeine groups... with Motrin being significantly more effective ($p = 0.03$) at the three-hour interval.

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Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory agents (see WARNINGS).

Warnings: Anaphylactoid reactions have occurred in patients with aspirin hypersensitivity (see CONTRAINDICATIONS).

Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. Motrin should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If Motrin must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin and the patient should have an ophthalmologic examination, including central visual fields.

Fluid retention and edema have been associated with Motrin; use with caution in patients with a history of cardiac decompensation.

Motrin can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin is added.

Drug interactions. *Aspirin:* Used concomitantly may decrease Motrin blood levels. *Coumarin:* Bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy nor by nursing mothers.

Adverse Reactions

Incidence greater than 1%

Gastrointestinal: The most frequent type of adverse reaction occurring with Motrin is gastrointestinal (4% to 16%). This includes nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of the GI tract (bloating and flatulence). **Central Nervous System:** Dizziness,* headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic:** Decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS).

*Incidence 3% to 9%.

Incidence less than 1 in 100

Gastrointestinal: Upper GI ulcer with bleeding and/or perforation, hemorrhage, melena. **Central Nervous System:** Depression, insomnia. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure. **Special Senses:** Amblyopia (see PRECAUTIONS). **Hematologic:** Leukopenia, decreased hemoglobin and hematocrit.

Causal relationship unknown

Gastrointestinal: Hepatitis, jaundice, abnormal liver function. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities. **Dermatologic:** Alopecia, Stevens-Johnson syndrome. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Hemolytic anemia, thrombocytopenia, granulocytopenia, bleeding episodes. **Allergic:** Fever, serum sickness, lupus erythematosus syndrome. **Endocrine:** Gynecomastia, hypoglycemia. **Cardiovascular:** Arrhythmias. **Renal:** Decreased creatinine clearance, polyuria, azotemia.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

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Do not exceed 2400 mg per day.

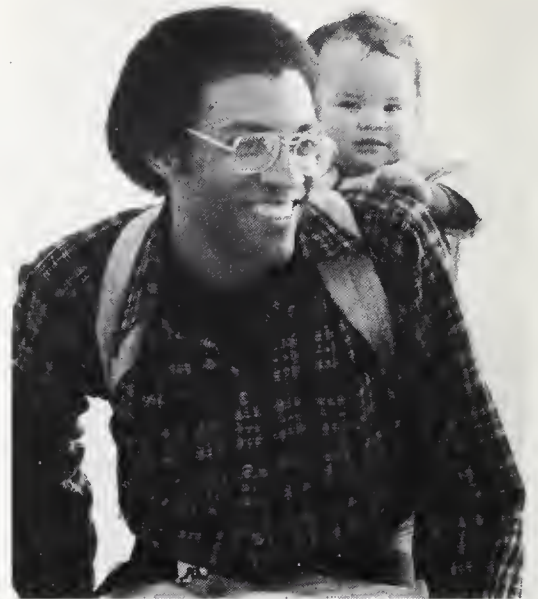
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AAFP Student Activities Fact Sheet

Continued the annual Hotline (which assists senior medical students unmatched by the National Intern and Resident Matching Program) to a year-round basis in order to facilitate students finding available residency positions . . .

Assisted in development of family practice residencies to reach an all-time high of 2536 first-year positions . . .

Continued to produce "Student Interest Kits," designed to provide information and helpful hints to aid students in establishing family practice special interest groups . . .

Continued to provide funding for speakers to address meetings of family practice special interest groups . . .

Expanded representation of medical student representatives (with voting privileges) to Academy commissions and committees . . .

Appointed voting delegates to Academy's Congress of Delegates, thereby providing students direct input into overall policy-making decisions . . .

Established a Committee on Minority Health Affairs . . . "to assess current efforts and to make recommendations on Academy programs and policies which encourage students of minority backgrounds to enter the field of medicine, particularly family practice." . . .

Published second "Guide to Family Practice Residency Programs," which provides pertinent information about each approved family practice residency program . . .

Continued to sponsor the annual National Conference of Student Affiliate Member Representatives in order to increase student involvement in AAFP activities . . .

Continued to foster Bylaws provisions at the AAFP constituent chapter level to provide for charter student affiliate component chapters . . .

Continued to represent the field of family practice/family physicians before various government committees and agencies regarding such issues as funding for family practice residency programs, physician reimbursement under Medicare and Medicaid, insurance, FDA regulations, etc. . . .

Continued liaison and participation in national meetings of the Student National Medical Association, the American Medical Student Association and the LaRaza Medical Association (LaRAMA) . . .

Continued to support student affiliate membership at the nominal annual fee of \$5.00, which provides all student members such tangible benefits as the monthly **American Family Physician** magazine and the monthly **AAFP Reporter** . . .

Continued to provide student members easy access to AAFP headquarters via the Academy's toll-free number—800-821-2512 . . .

Continued to waive registration fee for medical students attending the AAFP Annual Scientific Assembly, in addition to providing a student lounge (for informal conferences during the meeting) and a reception for students attending the Convention . . .

Continued to provide student affiliate members the opportunity to enroll in Academy-sponsored Group Life Insurance Plans at competitive premium rates and,

Continued bi-monthly "AAFP Resident" and Student Newsletter" in the Academy's monthly **AAFP Reporter**.

For additional information on any of the above programs, call AAFP headquarters today via the toll-free number—800-821-2512.

AAFP Activities Fact Sheet

Continued to support family physicians in their efforts to obtain hospital privileges for which they are qualified and trained . . .

Continued advocating a system for documentation of both the quantity and quality of family practice residents hospital experiences . . .

Expanded the computerized CME record-keeping system to 48 AAFP constituent chapters, involving 25,000 "active" members; in addition established a policy to provide the program to inactive and life members upon request at a nominal fee . . .

Continued representation of family physicians before various government committees and agencies on such matters as insurance, funding for family practice residency programs and physician reimbursement under Medicare and Medicaid, etc. . . .

Continued to produce the Home Study Self-Assessment, delivered to subscribers over a six-year period . . .

Established a research program in health care delivery, a research panel for clinical research and, began development of a bibliography of family practice research . . .

Began developing protocols to study the cost-effectiveness of delivery of medical care by family physicians . . .

Continued workshops for family practice educators, symposia on teaching skills and teacher development conferences as support measures for family practice residency programs . . .

Continued to provide workshops in the area of practice management and coping with practice pressures in addition to offering special workshops for the "physician coping alone" . . .

Began development of intensive membership recruitment plans geared toward women family physicians to solicit their involvement and input into Academy programming . . .

Co-sponsored National Conference on Rural Primary Care designed to provide physicians workshops and continuing education courses oriented toward rural health . . .

Continued to produce an Annual Scientific Assembly during which AAFP members may earn more than 30 Prescribed credits through ten program elements offered during the meeting . . .

Continued to provide members with additional CME programs through the monthly "Clinical Quiz", the **AFP Annual** and periodic clinical monographs in **American Family Physician** magazine . . .

Continued to provide AAFP members easy access to headquarters via the toll-free number—800-821-2512 . . .

Continued to offer members Academy-sponsored disability, life and retirement plans at competitive premiums . . .

Approved over 12,000 CME courses for AAFP credit . . .

Launched distribution program on guidelines of the latest and most effective cancer management techniques . . .

For additional information on any of the above, call AAFP today—toll free (800-821-2512).

New Bylaws

SDAFP members attending the Annual Business Meeting in Rapid City, August 14, 1981, will be asked to vote on a revised set of Bylaws. This will conform to changes made by AAFP, deleting the Constitution and incorporating it into uniform Bylaws suggested for constituent (state) chapters. A copy of the proposed Bylaws is on file at the state office for members, upon request.

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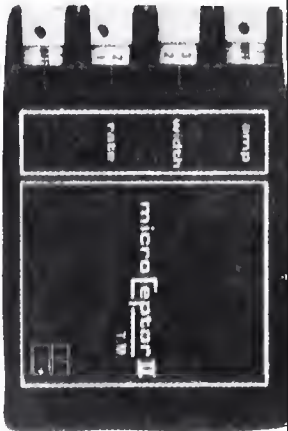
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A CENTENNIAL NOTE—

At the fourth annual meeting of the South Dakota State Medical Society held in Aberdeen on May 27, 1885, a movement for incorporation was initiated. Incorporation was finally attained at the annual meeting in Chamberlain in 1891. Increased interest in the Society was evidenced by the attendance of twenty-five members at the 1886 meeting held in Yankton. Scientific papers were read at this annual meeting including one on "Epidemic Fevers of Dakota" by Dr. F. Andros of Mitchell.

A CENTENNIAL NOTE—

In 1916 the Association created a Committee on Medical Defense to study the need for insurance for members to protect them in malpractice law suits. The Association contracted with the United States Fidelity and Guaranty Company of Baltimore, Maryland to provide insurance to members on an individual basis. Fifty-five doctors were individually protected. The Association obtained a group policy for professional liability with limits of \$10,000 to \$30,000 at an annual premium of \$25 per member participating.

S D

Future Meetings

May

Seminar for Ambulatory Surgery, Marriott Hotel, Chicago, IL, May 6, 7, & 8. Contact: Allan Higdon, 315 S. Ellis, Wichita, KS 67211. Phone: (316) 263-0124.

Eleventh Annual Meeting of the Great Plains Organization for Perinatal Health Care, Radisson South, Bloomington, MN, May 14-16. Contact: Virginia Rittenour, Coordinator, Box 50, 420 Delaware St., S.E., Minneapolis, MN 55455. Phone: (612) 373-5718.

Practical Dermatology in Primary Care, Mayo Mem. Aud., U of Minn., Minneapolis, Minn., May 15-16. 17 hrs. AAFP & AMA Category I credits. Fee: \$175. Contact: Cont. Med. Ed., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Proper Prescribing: Conflicting Signals, Mt. Sinai Sch. of Med., New York, NY, May 18-19. Category I credits. Fee: \$110. Contact: John P. Morgan, Sophie Davis School of Biomedical Ed., City Coll. of NY, Convent Ave. at 138th St., New York, NY 10031.

Topics and Advances in Pediatrics for Recertification, Mayo Mem. Aud., U. of Minn., Minneapolis, Minn., May 18-20. 18 hrs. AAFP & AMA Category I credits. Fee: \$150. Contact: Cont. Med. Ed., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Topics in Geriatric Medicine: Depression in the Elderly, Mayo Mem. Aud., U. of Minn., Minneapolis, Minn., May 21. 7 hrs. AAFP & AMA Category I credits. Contact: Cont. Med. Ed., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Medical Determinations in Worker Compensation, Ambassador West Hotel, Chicago, IL, May 21-22. Contact: A. Edward Doudera, J.D., Exec. Dir., Am. Society of Law & Medicine, 520 Commonwealth Ave., Boston, MA 02215. Phone: (617) 262-4990.

Otolaryngology Clinical Conference, Univ. of Iowa, Iowa City, IA, May 22. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa Coll. of Med., Iowa City, IA 52242.

Medical Directors Spring Meeting, Mayo Mem. Aud., U. of Minn., Minneapolis, Minn., May 22. 7 hrs. AAFP & AMA Category I credits. Contact: Cont. Med. Ed., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

International Symposium on Gilles de la Tourette Syndrome, Roosevelt Hotel, New York, NY, May 27-29. Contact: Sheldon Novick, M.D., TSA Med. Dir., Gateposts Fdn., 42-40 Bell Blvd., Bayside, NY 11361. Phone: (212) 631-0177.

Current Concepts in Radiation Therapy, Mayo Mem. Aud., U of Minn., Minneapolis, Minn., May 27-29. 20 hrs. AAFP & AMA Category I credits. Fee: \$250. Contact: Cont. Med. Ed., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Comprehensive Care of the Burn Patient, Hyatt Regency Hotel, Kansas City, MO, May 29-30. 13 hrs. Category I credits. Contact: American Burn Assoc., Robert Gillespie, M.D., 770 N. Cotner Blvd., #215, Lincoln, NE 68505. Phone: (402) 467-5454.

Workshop on Heart Attack Prevention, Spring Hill Ctr., Wayzata, Minn., May 31-June 2. 17 hrs. AMA Category I credits. Contact: Cont. Med. Ed., Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

June

John Lawrence Interdisciplinary Symposium on the Physical and Biomedical Sciences, Sioux Falls, SD, June 3-4. Contact: George P. Scott, Dept. of Chemistry, Univ. of SD, Vermillion, SD 57069.

Bariatric Surgery Workshop, Univ. of Iowa, Iowa City, IA, June 4-5. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa Coll. of Med., Iowa City, IA 52242.

Intensive Course in Pediatric Nutrition, Univ. of Iowa, Iowa City, IA, June 15-19. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa Coll. of Med., Iowa City, IA 52242.

July and August

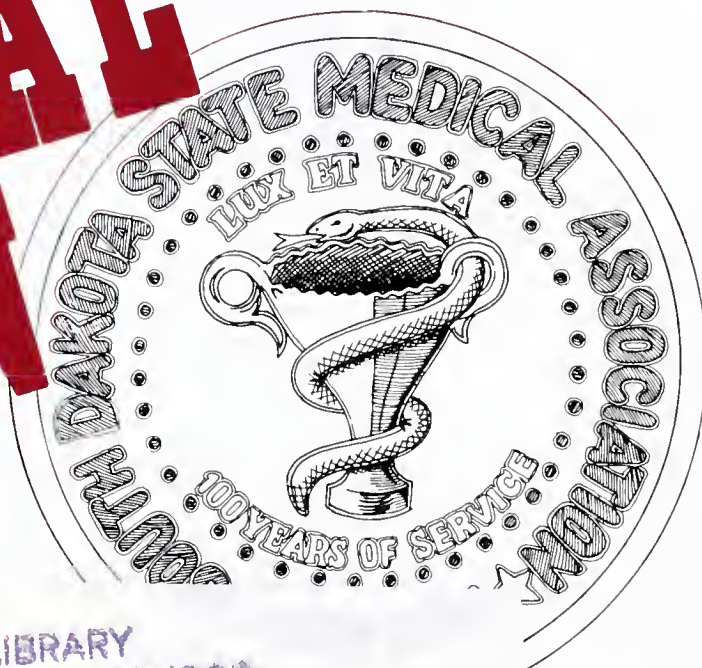
Summer CME Cruise/Conferences on Legal-Medical Issues, Caribbean Conference, July 29-Aug. 8, aboard TSS Fairwind. Mediterranean Conference, Aug. 22-Sept. 5, aboard Mts. Danae. Contact: International Conf., Suite C, 189 Lodge Ave., Huntington Station, NY 11746. Phone: (516) 549-0869.

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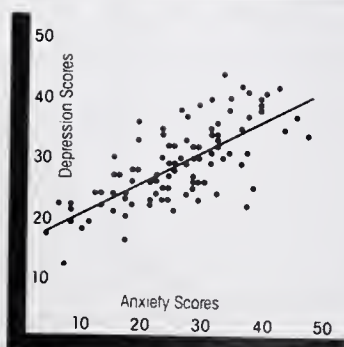
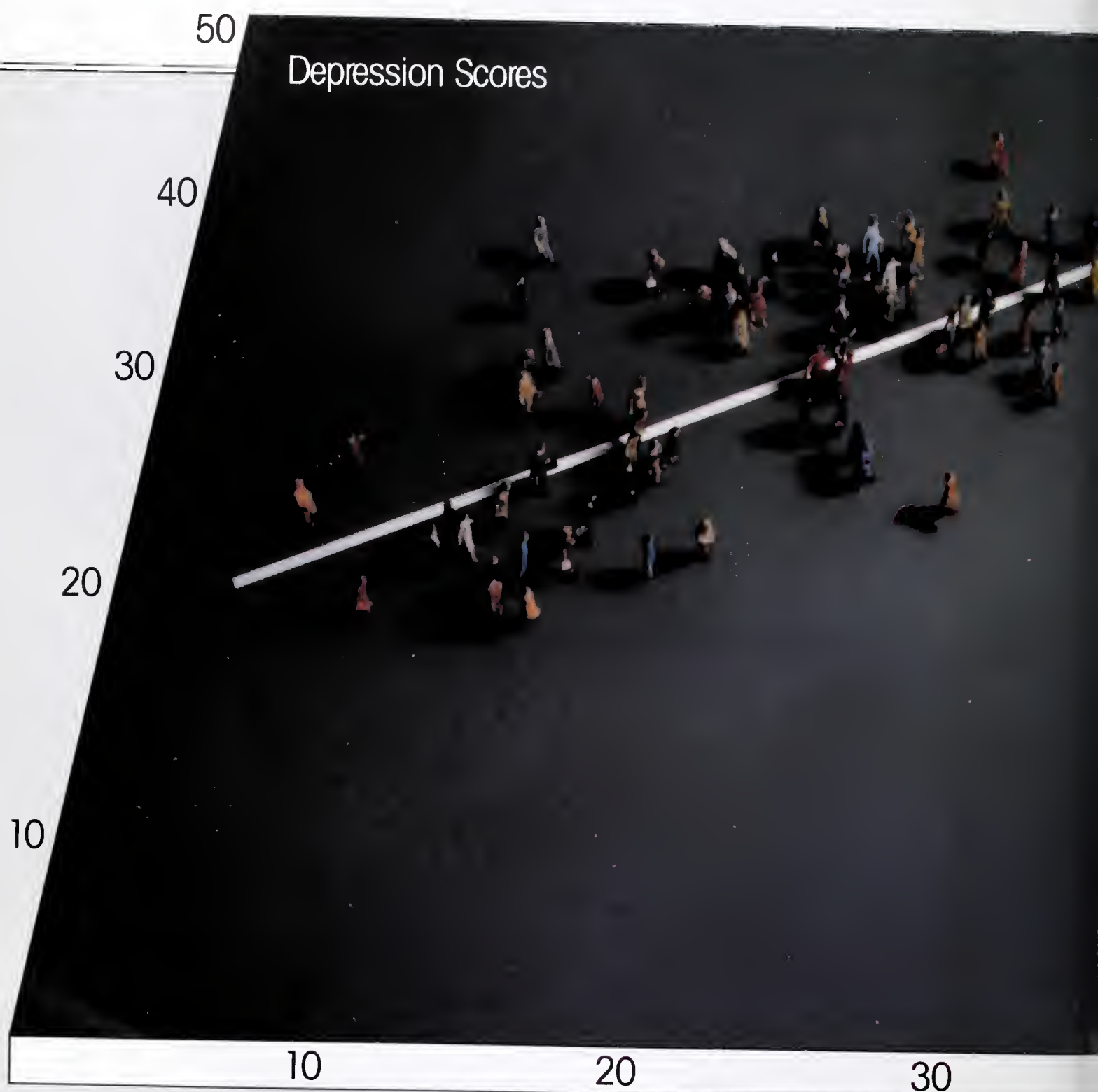


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FOR THE 7 OF 10 NONPSYCHOTI



Clear correlation between anxiety and depression³

The above graph illustrates a relationship between anxiety and depression, indicating that patients seldom present with anxiety or depression alone; more often they have both in varying degrees. Data based on a sampling of 100 outpatients (64 male; 36 female) seen at a general psychiatric clinic.

³Adopted from Coghorn, J. The anxiety-depression syndrome. *Psychosomatics* 11:438-441, Sept-Oct 1970.

DEPRESSED PATIENTS WHO ARE ALSO ANXIOUS^{1,2}

Most depressed patients are also anxious. . .

Some authors estimate that 70% of all nonpsychotic patients with symptoms of depression have concomitant symptoms of anxiety.^{1,2} One author found a distinct correlation between anxiety and depression scores in 100 nonpsychotic outpatients administered the Minnesota Multiphasic Personality Inventory in a general psychiatric clinic.³ As depression scores increased, so did anxiety scores. No attempt was made to select patients other than to exclude psychotics.

but not psychotic

The logic of treating both components of anxious depression is clear. Antipsychotics, like the phenothiazines, however, carry a well-documented risk of tardive dyskinesia.⁴ Because of this, an APA Task Force recently recommended the judicious use of phenothiazines in cases other than chronic psychosis or the use of alternative treatments.

A better way to give relief

Limbitrol combines the specific anxiolytic action of Librium® (chlordiazepoxide HCl/Roche)—a benzodiazepine with a long history of safe use—with the antidepressant action of amitriptyline, a tricyclic of established clinical efficacy. In comparison to phenothiazines, Limbitrol and its components have rarely been associated with tardive dyskinesia or other extrapyramidal side effects. And in terms of rapid response and patient compliance, Limbitrol appears to be superior to amitriptyline alone. Controlled multiclinic studies showed Limbitrol relieved more symptoms more rapidly than did amitriptyline.⁵ Despite a higher incidence of drowsiness, the dropout rate due to side effects was lower with Limbitrol. (See adverse reactions section in summary of product information on next page. As with any CNS-acting agent, patients should be cautioned about driving or using dangerous machines while on therapy with Limbitrol.)

References: 1. Rickels K: Drug treatment of anxiety, in *Psychopharmacology in the Practice of Medicine*, ed. Jorvik ME. New York, Appleton-Century-Crofts, 1977, p. 316. 2. Schatzberg AF, Cole JO: Benzodiazepines in depressive disorders. *Arch Gen Psychiatry* 35:1359-1365, 1978. 3. Claghorn J: The anxiety-depression syndrome. *Psychosomatics* 11:438-441, 1970. 4. The Task Force on Late Neurological Effects of Antipsychotic Drugs: Tardive dyskinesia, summary of a task force report of the American Psychiatric Association. *Am J Psychiatry* 137:1163-1172, 1980. 5. Feighner JP *et al*: A placebo-controlled multicenter trial of Limbitrol versus its components (amitriptyline and chlordiazepoxide) in the symptomatic treatment of depressive illness. *Psychopharmacology* 61:217-225, 1979.

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Please see summary of product information on next page.

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Before prescribing, please consult complete product information,
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Indications: Relief of moderate to severe depression associated with moderate to severe anxiety

Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.

Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.) Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving).

Usage in Pregnancy: Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage, withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline, symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).

Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated; sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12.

In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.

Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.

The following list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:

Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.

Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extropyromidal symptoms, syncope, changes in EEG patterns.

Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.

Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.

Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.

Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.

Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single h.s. dose may suffice for some patients. Lower dosages are recommended for the elderly. Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.

How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500, Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Packs of 50.

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THE ALUMNI ASSOCIATION

of the

UNIVERSITY OF SOUTH DAKOTA SCHOOL OF MEDICINE

The University of South Dakota School of Medicine Alumni Association was officially organized in 1980. It is a self-standing, incorporated organization. It is responsible for alumni programming and raising of funds for general support of the School of Medicine. The Association works closely with the South Dakota Medical School Endowment Association which provides funds for student loans and scholarships and for research support. Both organizations are administered by separate Boards of Directors with assistance from the School of Medicine.

As of 1977 the University of South Dakota School of Medicine is a four-year degree granting school, and through the Alumni Association the school, and past and present students will be better served.

Contributions may be sent to:

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Executive Proclamation

State of South Dakota

Office Of The Governor

WHEREAS, In 1981 the South Dakota State Medical Association is officially honoring the One Hundredth Anniversary of its founding; and,

WHEREAS, Physicians have been dedicated to providing competent medical service with compassion and respect for human dignity since the appointment of William Jayne, M. D., the first Governor of Dakota Territory; and,

WHEREAS, The South Dakota State Medical Association has had a beneficial influence on cultivating and advancing medical knowledge; elevating the standard of medical education; promoting the usefulness, honor and interests of the medical profession; enlightening and directing public opinion in regard to the duties, responsibilities and requirements of physicians; and facilitating and fostering friendly transactions among those who are engaged in it:

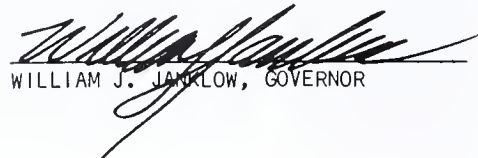
NOW, THEREFORE, I, WILLIAM J. JANKLOW, Governor of the State of South Dakota, do hereby proclaim May 28 through 31, 1981, as

SOUTH DAKOTA STATE MEDICAL ASSOCIATION CENTENNIAL

In South Dakota.



IN WITNESS WHEREOF, I have hereunto set my hand and caused to be affixed the Great Seal of the State of South Dakota, in Pierre, the Capital City, this Twenty-sixth Day of February, in the Year of Our Lord, Nineteen Hundred and Eighty-One


WILLIAM J. JANKLOW, GOVERNOR

ATTEST:


ALICE KUNDERT, SECRETARY OF STATE

The South Dakota State Medical Association



The first executive office of the South Dakota State Medical Association was in the First National Bank Building from 1946-1960. John Foster was hired as the first executive secretary in 1946, a position he held until 1964.





In 1960 a new executive office was built at 711 North Lake Avenue, Sioux Falls. Richard C. Erickson replaced John Foster as executive secretary in 1964 and remained until 1972.



The State Association built a second office at 608 West Avenue, North, Sioux Falls in 1974. In 1972 Robert D. Johnson became executive secretary and remains in that capacity at this time.

Aberdeen District Medical Society (#1) **1957-1981**

Marie Hovland*

In 1957 St. Luke's Hospital, Aberdeen, received a Ford Foundation grant of \$86,400 for renovation and additional facilities. St. Luke's was established in 1901 by the Presentation Sisters. It has grown from a small 15 bed building to a modern up-to-date complex accommodating 160 patients plus 20 bassinets.

1958. September. Earle A. Pittenger, M.D., died. Dr. Pittenger was district councilor from 1928-35, District I president in 1934 and South Dakota state president in 1938.

1959-60. G. J. Bloemendaal, M.D., Ipswich, was the ninth president of the S. D. Academy of Family Physicians. Charter date was January 1, 1951 with F. F. Pfister, M.D., Webster, president.

1963. May. Dr. and Mrs. B. C. Murdy, long-time residents of Aberdeen, celebrated their 50th wedding anniversary.

1963. September 14. Jim Berbos, M.D. (40) assisted by B. F. King, M.D., and A. J. Janusz, M.D., delivered and cared for the Fischer quintuplets. The four girls and one boy were the first surviving quints in this country. The event focused international attention on Aberdeen. Dr. Berbos began an ophthalmology residency in 1964 and has since established his practice in California.

1963. September. R. D. Alway, M.D., was made an honorary life member of district I and SDSMA. Dr. Alway was the first president of St. Luke's Hospital medical staff in 1915 and in 1920 was SDSMA president.

1963. November. G. J. Bloemendaal, M.D., was recognized by the Ipswich community, where he resides at this time, for his 35 years of medical service.

1966. Paul R. Leon, M.D., and Walter Miller, M.D., received the first Aesculapius Award presented by the SDSMA at a state meeting for an exhibit entitled, "Blow-Out" Fractures of the Orbit.

1966. Paul G. Bunker, M.D., received the Chevalier Jackson Award of the American Broncho-Esophogological Association for outstanding achievements in broncho-esophogology at their annual meeting in Puerto Rico. Dr. Bunker was an active member of the association since 1944 and chairman of that group's Committee for the Prevention of Foreign Body Accidents. In 1970 Dr. Bunker was elected to life membership in District I and the SDSMA. He died in September, 1971 at the age of 66 following an extended illness. Dr. Bunker practiced in Aberdeen 40 years.

1968. G. J. Bloemendaal, M.D., Ipswich, received the Distinguished Service Award at the 1968 annual state meeting in Aberdeen.

1969. July. District I reached a medical milestone with the opening of a 34-bed wing at the new Dakota Midland Hospital. Since then additions have been made. Health Central, Inc. assumed operation in 1974.

1972. Radiologist Paul V. McCarthy, M.D., retired and was awarded life membership in the district and SDSMA. He died April 29, 1977 in Aberdeen at age 77. Dr. McCarthy was also a 50 year club member.

1972. December 15. B. F. King, M.D., age 52, was struck and killed in a highway accident near Mitchell. Dr. King was on the S. D. Blue Shield Board of Directors for many years. At the 1973 state meeting William Taylor, M.D., presented the SDSMA presidential medallion to be passed on to each succeeding president. Also in 1973, Dr. King posthumously received the SDSMA Community Service Award.

*Member, Centennial Committee and South Dakota Medical Auxiliary, Aberdeen, SD.

1972. May. The District I Medical Association contributed \$500 to District I Auxiliary for the purchase of a VASC audiometer to be used for hearing screening in conjunction with the Fortunate Four Pre-School Medical Survey of Vision and Hearing. The Auxiliary appreciated this generosity.

1973. William R. Taylor, M.D., Aberdeen, was elected SDSMA president.

1974. October 19. Loyola Donely Taylor, M.D., Aberdeen, wife of William R. Taylor, M.D., died unexpectedly in a Rochester, Minnesota hospital.

1974. J. D. Alway, M.D., Aberdeen physician from 1925-63 died at age 78 in Sun City, Arizona. In 1963 he was granted honorary life membership in SDSMA. He served on the Council of SDSMA, the Board of Basic Science Examiners and was involved in various community projects. In 1920 Dr. Alway was SDSMA president.

1974. Rudolph Avotins, M.D., was awarded honorary membership in District I and SDSMA.

1976. January. B. C. Gerber, M.D., was appointed by Governor Richard Kneip to a three year term on the South Dakota Board of Basic Science Examiners.

1976. June. Ed A. Rudolph, M.D., retired from his 40+ years in the practice of ophthalmology in Aberdeen. He and his wife Beulah now reside in Woodland Hills, California. The Rudolphs celebrated their 50th wedding anniversary before leaving Aberdeen.

1976. October 4. John L. Calene, M.D., age 81, died in Costa Mesa, California. He established practice in Internal Medicine in Aberdeen in 1925 and retired in 1975. Dr. Calene was the first diplomat of the Board of Internal Medicine in South Dakota and was past president of SDSMA in 1949. He was also a member of the American College of Physicians and American College of Cardiology.

1976. March. E. A. Rudolph, M.D., and J. L. Calene, M.D., were named honorary life members of the SDSMA.

1977. James Hovland, M.D., Al Janusz, M.D., and John Rodine, M.D., were selected as a steering committee for St. Luke's Hospital to establish a hospital sponsored group medical practice which will offer general medical care to entire families, made possible by a grant from the Robert Wood Johnson Foundation.

1977. W. R. Taylor, M.D., Aberdeen, was South Dakota AMA Delegate. His appointment extended for two terms which expired in 1981.

1978. December. The Family Health Center, affiliated with St. Luke's Hospital, opened. Carson B. Murdy, M.D., was the first physician there to practice general/family medicine. The St. Luke's Hospital Educational center was designated as the Carson B. Murdy, M.D., Education Center in honor of Dr.

Murdy's many years of service to the hospital and Aberdeen community.

1978. April. Susan M. Ostrowski, M.D., Eureka, SDAFP member, was named Diplomat of ABFP having successfully completed the two day written exam. Susan's husband, Peter, is an attorney in Eureka and a member of District I Auxiliary.

1978. February. M. R. Gelber, M.D., retired Aberdeen physician, died in Scottsdale, Arizona.

1978. July. Agnes Keegan, M.D., former Aberdeen physician from 1936-67 died May 17 in Oak Park, Illinois. She was an honorary member of the Aberdeen District and State Medical Association.

1979. January. Jerome Eckrich, M.D., was honored by Creighton University and the Alumni Association for outstanding service. He was recognized for his 50 years of service and for his loyalty to Creighton University. He is a 1928 graduate of Creighton.

1979. April. The SDSMA Council voted to invite the Auxiliary to name representatives to attend the meetings of the Commission on Internal Affairs, Communications and Liaison and the Commission on Legislation and Governmental Relations.

1979. June. G. J. Bloemendaal, M.D., Ipswich, received the SDSMA 50 year award.

1979. June. AMPAC Award to SoDaPAC for being the top PAC in the country in the category of contribution per member.

1979. August. The St. Luke's Hospital educational center in Aberdeen was designated as the Carson B. Murdy, M.D., Educational Center during a special meeting of the medical and dental staff. This designation was made in honor of Dr. Murdy's many years of service to the hospital and the community of Aberdeen.

1980. April. W. R. Taylor, M.D., Aberdeen, has been appointed to the Northern State College Foundation Board of Directors.

1980. W. R. Taylor, M.D., was elected president of the North Central Medical Conference for 1980-81. A national honor for District I. Dr. Taylor has also been actively involved in AMPAC and SoDaPAC for many years.

1980. March 18. Carson B. Murdy, M.D., Aberdeen, died at the age of 64 following an extended illness. Dr. Murdy served overseas with the medical corps during WWII and returned to Aberdeen in 1945 to establish his medical practice. Dr. Murdy taught scuba diving throughout the state, was Northern State College physician for many years and was active in Boy Scout programs in the Aberdeen community.

1980. May. Winston Odland, M.D., Aberdeen, was elected president of the SDSMA for 1980-81.

Watertown District Medical Society (#2)

C. Rodney Stoltz, M.D.*
Virginia Stoltz**

From Watertown's beginning in 1879, it is evident that physicians and their wives provided early community leadership in the business, governmental, and cultural affairs of the area. Dr. Hans Martin Finnerud was one of the founders of the Midland National Life Insurance Company in Watertown in 1906, and served a term in the South Dakota Senate. He and Dr. R. F. Campbell were pioneer physicians and operated the first hospital in Watertown.

One of the oldest hospitals in South Dakota was Luther Hospital, established in Watertown in 1909 by an organization from the Lutheran Churches. (It became Memorial Hospital, a community facility, in 1951.) In 1911 Dr. H. J. Bartron, Sr. opened the Bartron Hospital, and in 1950 St. Ann Hospital was opened by the Bernardine Sisters, O.S.F. of Reading, Pennsylvania. With the hospital in Clear Lake, these facilities have provided the area with excellent medical care.

Since the early years, district physicians have been active in city government, the Chamber of Commerce, service organizations, and business enterprises. They have been leaders in allied health organizations and agencies, as well as in their professional groups. In late years, Dr. G. Robert Bartron of Watertown served as state senator.

It is not known precisely when the District Medical Society organized. At a meeting in 1931 the members extended a vote of thanks to Dr. J. B. Vaughn of Castlewood for writing a complete history of the Society. It was ordered a copy be placed in the permanent records, but it cannot be found today.

The earliest minutes now available begin in 1928.

* Retired obstetrician-gynecologist, Watertown, SD.

**Past President, State and American Medical Auxiliary, Watertown, SD.

They include a copy of a letter to each member urging attendance at a special meeting with local members of the state legislature. Of great concern was the need for a basic science law to protect the public from medical quacks and cults, but it was many years before such a law was enacted. Dues were \$10, raised to \$15 in 1930, and there were more than 20 on the roster. In these early years physicians from the Whetstone Valley District attended meetings in Watertown. Throughout its history the district has provided excellent programs and distinguished speakers for the profession.

As far as can be ascertained, most of the time 100 percent of physicians in the district have joined the district, state and national associations, and a good many have supported medicine's political action groups, SoDaPAC and AMPAC.

There was considerable trouble in getting a suitable set of bylaws drafted and adopted. Bylaws committees would present ideas, then came revisions, samples from other districts would be sought, and the document was several years in the making.

There were many stormy sessions at meetings. One secretary noted that a "discussion of Blue Cross plan ended in a riot." The ethics of professional advertising caused problems; there was division on whether to invite Auxiliary members to have dinner with the Society prior to their meetings, the Farmer's Aid Corporation medical plan caused dissent, etc. There was unity, however, in the constant battle to stave off comprehensive socialized medical care, and finally, good support for promotion of a four-year medical school. For the most part district members supported the projects and efforts of the State Medical Association and assumed leadership roles.

From early in its existence the Society has pro-

moted public health education and performed community services. The group sponsored radio programs and television broadcasts on health matters; executed immunization programs; provided physical examinations for school athletes, Scouts, etc.; provided funds to place **Today's Health** in schools, libraries, and hospitals; sponsored essay contests; and at one time formed a speakers' bureau. Cancer symposiums were sponsored in cooperation with the American Cancer Society, and conclaves of physicians and dentists were held. The Society urged physicians to serve as coroners. In 1943 the Society purchased three \$100 war bonds.

Nine members of the Watertown District Medical Society have served the South Dakota State Medical Association as president. The first was Dr. Edwin T. Ramsey of Clark, who was president in 1906-07. Then followed H. M. Finnerud of Watertown in 1910-11; J. B. Vaughn of Castlewood in 1915-16; William G. Magee of Watertown in 1934-35; H. Russell Brown of Watertown in 1947-48; C. Rodney Stoltz of Watertown in 1960-61; John J. Stransky of Watertown in 1967-68; G. Robert Bartron of Watertown in 1971-72; and Gerald E. Tracy of Watertown in 1975-76. In addition, Dr. Brown served for many

years as the State Association's delegate to American Medical Association meetings and Dr. Tracy is at present the AMA delegate. Dr. Stransky served as an alternate delegate. Dr. Brown also served the AMA as a member of the Commission to Study Prepaid Medical Care Plans.

Watertown was the site of nine state conventions: 1906, 1914, 1919, 1923, 1932, 1940, 1945, 1965, and 1969. At the 1965 meeting, the Association hosted the AMA president, Donovan Ward; the AMA Auxiliary president, Mrs. W. H. Evans; and the president of the American Academy of General Practice, Amos Johnson. It marked also the first joint luncheon and program of the Medical Association and its Auxiliary. The program was on Medicine and Religion, and clergymen in the area were invited as guests. Another AMA president visited Watertown in 1963: Dr. Edward R. Annis was invited by G. Robert Bartron as president of the Chamber of Commerce to speak at the Chamber's annual meeting. Dr. Annis visited the Fischer quintuplets in Aberdeen during that visit.

The Watertown District Medical Society takes pride in the part it has played in 100 years of Dakota medicine.



H. M. Finnerud, M.D., one of Watertown's first physicians, was also an enthusiastic hunter and is shown here on a duck hunting trip to the Chain Lakes area northwest of Watertown in 1910 or 1911 aboard a Buick of that period.

Reprinted from Watertown Public Opinion, Wednesday, June 6, 1979.

Synopsis Of The Brookings-Madison District Medical Community (#3)

Myron C. Tank, M.D.*

Note: The compilation of a history of the Third District Medical Society would be unproductive; therefore, it has been decided to compile a synopsis of the medical community. Acknowledgment should be made of the Third District's honor of having the 1981-82 president of the SDSMA, Dr. Bruce Lushbough, as a member.

In 1900 three physicians were in Brookings, Drs. Collier, Miller and Parsons. The first hospital was built in 1907 and served Brookings until 1936 when the Brookings Municipal Hospital was constructed. The present Brookings Hospital was completed in 1964 and is presently undergoing additions. The Hospital Auxiliary was organized in 1954.

In the 1920's the Brookings Clinic was formed with Dr. Stoll, Warner and Tillisch. In 1929 the clinic was comprised of Drs. Tillisch, Miller and Davidson. The medical community also consisted of Drs. Gulbrandsen, Whitehead, Kellogg, Ingelson and Tank. A new Brookings Clinic was built in 1956 which served until 1974 when the present Brookings Clinic was completed. Today the clinic houses Drs. Robert Henry, Bruce Lushbough, Ronald Long, Walter Patt, Charles Roberts, Robert Shaskey, Curtis Wait and Richard Wake. Brookings has been fortunate to have Dr. Saul Friefeld as an itinerant radiologist.

The Watson Clinic was opened by Dr. E. S. Watson in 1939. Dr. C. A. Kershner, an ophthalmologist, began practice there in 1940. The clinic closed in 1972 with the retirement of Dr. Watson.

The Yorkshire Eye Clinic was built in 1980 consisting of two ophthalmologists, Drs. R. R. Tesch and S. J. Bandiera.

The first recorded doctor to arrive in Flandreau was in June 1878, when Dr. J. A. Scaman began his

practice. Over the years many physicians have served the area; however, it would be remiss not to mention Dr. F. A. Spafford who served the community from 1884 to 1922 and after whom the present grade school is named. Others who have practiced since 1900 are: Drs. Rider, Fisk, Mueller, Boyd, Benjamin, Hurewitz, Shaw and Klar. At present, Flandreau is served by Drs. B. T. Otey and D. P. Warlick.

Hospital care was available in a home in the south part of town before 1900. In 1914 or 1915 a three-story house was converted to a hospital which served the community until 1937 when the present hospital, built with P.W.A. labor, opened to receive patients.

In 1878 Old Madison has records of its first doctor, Dr. A. E. Clough. Later he moved his practice to New Madison where he practiced in the old hospital until 1902. That year the Clough Hospital came into existence. A new Madison Hospital (now Dakota State College's Heston Hall) opened in 1920 and served the community until 1963 when the present Madison Community Hospital was completed.

Many doctors have practiced in Madison during these years including Drs. Clough, Duff, Daniels, Jenkes, Fruenfeld, Hoaglund, R. S. and J. R. Westaby, D. S. Baughman, Kellogg, Allison, Goldman, Files, Willoughby of Winfred, Jordan of Chester, Hovde and more recently, Drs. Whitson, Sherwood, Wold, Lillard, Muggly, Hillan, R. Baughman, Wier, Anderson, Belatti, Reagan,

*Retired Physician, Brookings, SD.

Stensrud, Karlen, Lampert, Lavarro, Edwards, Appelwick and Wilde. In the past 32 years, Madison has been fortunate to have the services of Dr. S. Friefeld of Brookings as radiologist for two days each week.

The first doctors to practice in Volga were Drs. D. L. Scanlan and E. T. Torwick. Dr. Alonzo Peeke joined them in April 1929. The Volga Hospital was started by Drs. Scanlan and Torwick with the first facilities being five beds above the Drug Store in 1905. A sixteen-bed hospital was constructed on the old school grounds in 1909. In 1964 the hospital was closed because it could not meet the strict State standards without expensive changes. At that time we were invited and joined Brookings in building a hospital.

Acknowledgment

Gratitude is expressed to Miss Gertrude Young, English Department, SDSU; Mrs. Mary C. Galbraith; and Drs. B. T. Otey, Flandreau, H. Stensrud, Madison and A. Peeke, Volga.

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Huron District Medical Society (#5)

David J. Buchanan, M.D.*

It would be easier to write a Christmas letter for the Seven Dwarfs than the Centennial history for a medical society. Perhaps this short piece explains why Dr. O. M. Farrington was present in Huron, Dakota Territory when the surveyor's stake was driven into the muddy, April 1880 ground. It was written by a grandson of a 58 year District V member who did know why he settled in Beadle County.

Raised on a plain.
 Bright sun, I learned to shield the eye.
 Shyness, pain
 leads to the hollow scratch against a toad's back
 a wisely silent teacher.
 Quiet and hear
 listen not to follow.

On a flat you can see.
 Like being on your back you can see.
 Distance, no wall but perception
 in the open you can find more.
 A field is green then brown.
 Time in color
 color in the black earth wet with white spring snow.

It is raining as I slept.
 The plains close touched me in surround.
 Open, they surround me now.

D.M. Buchanan

This poem may also explain why 15 of the present 29 names on the District V roster are "home grown" pioneers in medicine. Perhaps 200 names could be tabulated between Farrington and Dr. Stephen Schroeder (Miller, 1980). Don't leave anyone out, they remind you. Seven state presidents you are told: Moody (1886), Grosvenor (1930), Shirley (1940), Saxton (1950), Buchanan (1960), Hohm (1966), Leigh (1977). How many 50 year pins span the years? Allopaths, homeopaths, osteo-

paths, all contributed to the community welfare beyond their office doors. How many towns of 500 souls traded their several doctors for better roads?

Would you like to have heard the gynecological paper presented by Dr. Dickinson to the Central South Dakota Society meeting in Pierre May 7, 1884? Dr. George Moody, a railroad surgeon from Huron also spoke. Perhaps he told how one day 700 settlers landed in Huron via the Chicago and Northwestern RR trains.

In 1885 nine physicians were licensed in Beadle County for the 500 inhabitants. Crop failures and hard times slowed down the migration in the 1890's. Dr. T. J. Wood practiced in Huron from 1890 till 1953 and built one of the first hospitals. (Ten beds in a two story frame house—still standing)

Dr. B. A. Bobb of Mitchell signed the Huron District charter June 1, 1904, and listed Dr. Charles Lavery as secretary and Dr. Wm. Lane as president. The modern hospital history of the area continued with the opening of the Sprague hospital with 57 beds in 1915. Dr. J. S. Tschetter opened his Samaritan hospital in 1923. Surgery moved from the kitchen table to the operating room.

The 1930's and little grew and much blew but the physicians dug in. In the 1940's the district sent its doctors off to the Service as they had in 1917 . . . Dean, Adams, Saylor, Hohms, Buchanan. The first unified hospital staff in Huron followed the war with the opening of St. John's building in 1946. Soon new hospitals in Miller and DeSmet developed as new men came in.

These years mirrored the changes in practice with new physicians coming in as specialists to join family practitioners. In 1974 we took in Dr. Lawrence Betts, our first osteopath member. The 1974-1976 court battle over hospital By-Laws unified the District, the State Association and the AMA. The local involvement and legal victory culminated in the

*Family Physician, 707 Dakota Ave., S., Huron, SD.

purchase of the Huron Regional Medical Center in 1978. About this time a new Dr. Dean and new hospital began functioning in Wessington Springs. A new building program for the Huron Staff is planned for the Centennial year.

Two hundred names, bald heads and bearded

faces, have practiced here. There were midwestern names like Hanson, unfamiliar European ones such as Avotins and Jacoby, and new ones from the Orient.

“The plains close touched me in surround
Open, they surround me now.”

FEE BILL
OF THE
Huron District Medical Society
ADOPTED JANUARY 1, 1910

MEDICINE					
Ordinary day visit to residence	\$	\$ 2.00	Reducing dislocation of elbow	25.00 to	50.00
Mileage: \$1.00 per mile; and \$2.00 in addition to it for the visit			Reducing dislocation of wrist	15.00 to	25.00
Night visits, from 9:00 p.m. to 7:00 a.m. ...		3.00	Reducing dislocation of other joints	5.00 to	25.00
Each additional patient in family		1.00	Old and munited fractures and unreduced dislocations, double the fee for that of recent cases.		
Emergency calls		3.00	Operation for strabismus	50.00 to	100.00
Visit to contagious diseases, i.e., scarlet fever, diphtheria, small pox, and septicemia		3.00	Operation for cataract	100.00 to	150.00
Consultation, at office or phone	1.00 to	10.00	Operation for iridectomy	75.00 to	150.00
Vaccination, at office		1.00	Operation for harelip	50.00 to	150.00
Opinion involving legal questions	25.00 to	100.00	Operation for cleft palate	100.00 to	200.00
Post-mortem examination	25.00 to	50.00	Extraction of foreign body from eye, ear, nose, pharynx esophagus	1.00 to	50.00
Antitoxin, for each injection, in addition to visit		1.00	Removal of nasal polypus	10.00 to	25.00
Two or more patients living in the same neighborhood shall be charged as separate visits.			Removal of adenoids	15.00 to	50.00
Urinalysis, chemical examination		1.00	Tonsilectomy	15.00 to	50.00
Microscopical examination		5.00	Excision of uvula	5.00 to	10.00
Office treatment of uncomplicated gonorrhea, first treatment, in advance		10.00	Use of stomach pump in poisoning	10.00 to	25.00
Office treatment of syphilis	5.00 to	50.00	Tracheotomy	50.00 to	100.00
Examination for insurance	2.00 to	10.00	Intubation	25.00 to	50.00
Attendance at court, per diem	25.00 to	50.00	Reducing hernia by taxis	10.00 to	25.00
Consultation fee	10.00 to	25.00	Operation for strangulated hernia	75.00 to	150.00
SURGERY			Application of truss	1.00 to	5.00
Amputation of thigh	\$ 75.00 to	\$150.00	Operation for fistula in ano	50.00 to	100.00
Amputation of leg	50.00 to	100.00	Operation for vesico or recto-vaginal fistula		100.00
Amputation of fingers or toes	10.00 to	20.00	Operation for hemorrhoids	35.00 to	75.00
Amputation of arm	50.00 to	100.00	Operation for imperforate anus	50.00 to	100.00
Amputation of penis	25.00 to	50.00	Operation for prolapse of rectum	50.00 to	100.00
Fracture of femur	50.00 to	100.00	Tapping for hydrocele	5.00 to	10.00
Fracture of leg	35.00 to	100.00	Radical operation for hydrocele, injection method	20.00 to	30.00
Fracture of ribs	5.00 to	25.00	Radical operation for hydrocele, open operation	35.00 to	75.00
Fracture of arm or forearm	25.00 to	75.00	Introducing catheter	2.00 to	10.00
Fracture of lower jaw	25.00 to	100.00	Operation for phimosis, paraphimosis or circumcision	15.00 to	50.00
Fracture of clavicle	25.00 to	50.00	Examination for stricture of urethra		5.00
Fracture of small bones	10.00 to	75.00	Operation for stricture of urethra	20.00 to	50.00
All compound or comminuted, 50 per cent extra.			Examination for calculus in bladder	5.00 to	10.00
For each subsequent dressing of fracture of bone	\$ 3.00 to	\$ 10.00	Lithotrity	50.00 to	100.00
Reducing dislocation of hip	30.00 to	75.00	Lithotomy	100.00 to	300.00
Reducing dislocation of knee	25.00 to	100.00	Castration	25.00 to	50.00
Reducing dislocation of ankle	15.00 to	25.00	Paracentesis thoracis or abdominis	10.00 to	50.00
Reducing dislocation of shoulder	15.00 to	50.00	Operation for appendicitis	100.00 to	150.00
			Operation for radical cure of hernia, single	50.00 to	100.00
			Operation for radical cure of hernia, double	75.00 to	100.00
			Application of plaster jacket	10.00 to	25.00

Trephining cranium	50.00 to	100.00
Operation of resection and necrosis	50.00 to	100.00
Opening abscess, at office	1.00 to	5.00
Ovariectomy or other abdominal section ..	100.00 to	200.00
Extripation of tumors in dangerous localities	50.00 to	100.00
Extripation of other tumors	5.00 to	50.00
Removal of breast	75.00 to	200.00
Ligation of principal arteries	10.00 to	75.00
Venesection		5.00
Tenotomy	25.00 to	30.00
Operation for club foot	50.00 to	100.00
Aspirating joints	5.00 to	10.00
Operation for bunions	50.00 to	100.00
Operation for ingrowing toe nail	10.00 to	25.00
Short anesthetic of less than one hour		5.00
Long anesthetic		10.00
First assistant in major operations		25.00

GYNAECOLOGY

Primary examination of uterus and appendages or rectum	\$ 2.00 to	\$ 10.00
Subsequent examination and treatment ..		1.50
Removal of uterine polypus	10.00 to	25.00
Operation for lacerated cervix uteri	25.00 to	50.00
Perineorrhaphy	25.00 to	50.00
Alexander's operation		100.00

Operation for imperforate hymen	5.00 to	20.00
Curettement of uterus	25.00 to	50.00
Dilating cervix for dysmenorrhea or sterility	10.00 to	50.00

OBSTETRICS

Normal labor not over four hours duration	\$ 25.00
For extra detention, in all cases, each hour or fraction thereof	1.00
Instrumental, protracted or complicated cases; according to condition.	
Miscarriage	25.00 to 100.00
Craniotomy	50.00 to 100.00
Caesarean section or symphysiotomy	50.00 to 100.00
Caring for third stage labor half regular fee	

BACTERIOLOGY

Examination of sputum for tubercle bacilli	\$ 5.00 to	\$ 25.00
Throat culture	5.00 to	15.00
Blood examination	1.00 to	10.00
Items of service not specially enumerated in this Fee Bill shall be charged in proportion to the nature, extent and importance of service.		
All accounts shall be considered due when service is rendered.		
It shall be deemed sufficient cause for the expulsion of any member of this society to attend families by the year, or to make any bargain or arrangement, the tendency of which is to avoid the spirit and effect of the above list of charges.		



Dr. Robert Buchanan, long-time Huron physician, driving his father and mother through an Iowa town in 1913.

showed up; covers had been laid for 28. There is no record of any resignations.

In 1903 the name of the Society was changed to the Seventh District Medical Society. Twenty-two names were listed as members but places of residence not given. One of the first actions of the newly named Society was to curtail newspaper advertising. In 1906 the membership dues were raised to \$5, but this included State Association dues. In 1907 the Seventh District spent \$219.65 to entertain the State Society at their annual meeting. In April 1912 an epidemiologist from Minneapolis spoke at a regular meeting. This was the District's first attempt to bring in outside speakers.

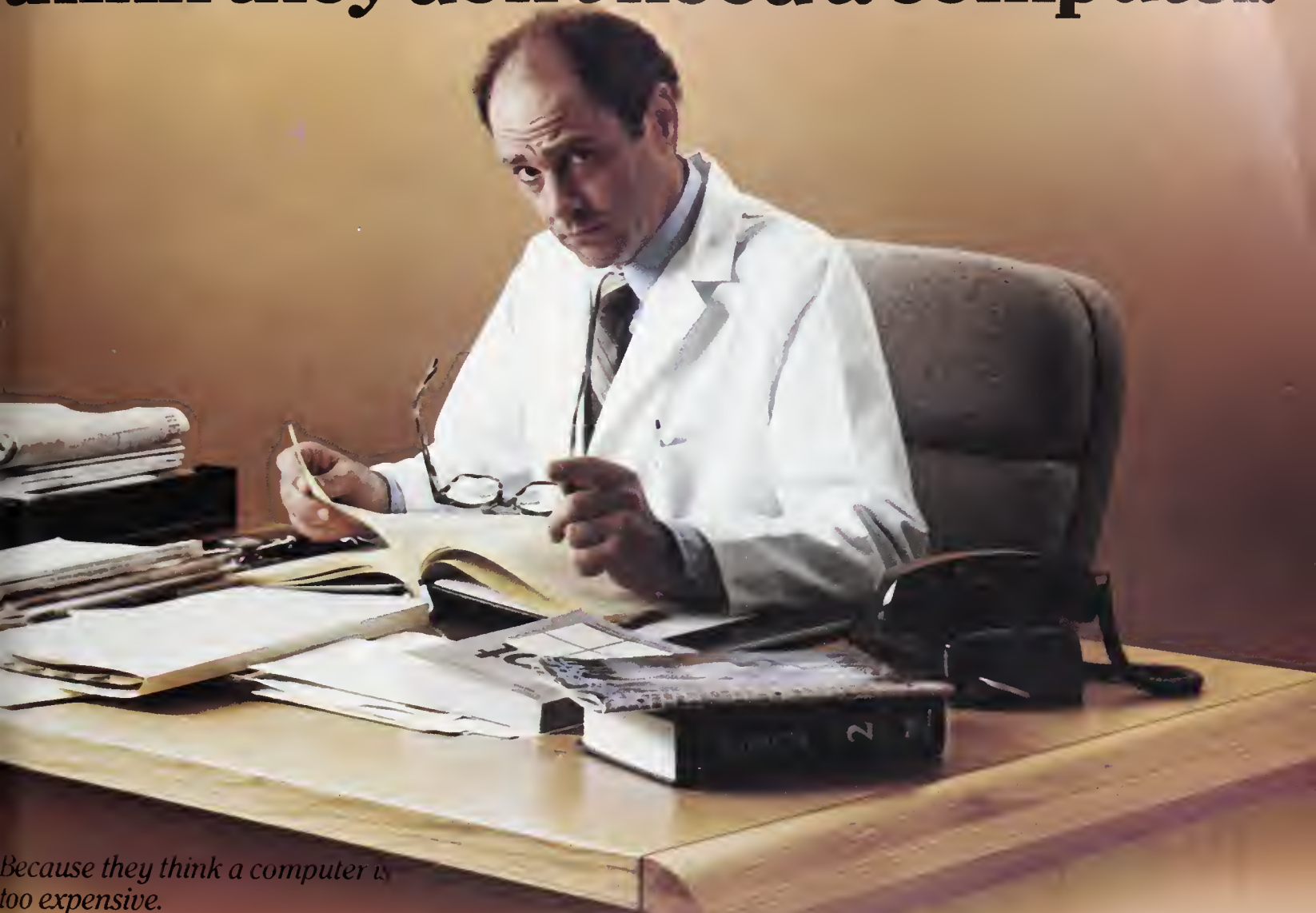
In 1917 some of the District members went into uniform as medical officers in World War I. There is no record of the number, names or rank held of those who enlisted. Early in 1918 the District voted

to treat soldiers' dependents free of charge until the end of the war. By the early 1920's the District had 62 members. During the depression of the 1930's not more than ten or twelve doctors came into the District. In the early World War II years a few doctors were called into service as they were in the Reserves. South Dakota had so few physicians that the doctors' draft wasn't enforced in the state. After the War doctors in large numbers came into the District, especially specialists. Early in 1954 the Seventh District petitioned the State Association to look into the feasibility of a Blue Shield plan for the entire State. This was done and we have had a statewide Blue Shield plan since 1956. In 1980 there were 198 members of the District. There has been only one doctor in the district convicted of a crime. He was one of the founding members in 1883. He served a term in the Minnesota Penitentiary for criminal abortion; date unknown.



South Dakota had the first Interallied Professional Association in the United States. Organizers of this Association included from left to right: W. E. Donahoe, physician; Roy Rooney, druggist; Tom Kinsred, veterinarian; W. P. Henderson, dentist; C. M. Austin, hospital administrator; Agnes Thompson, nurse.

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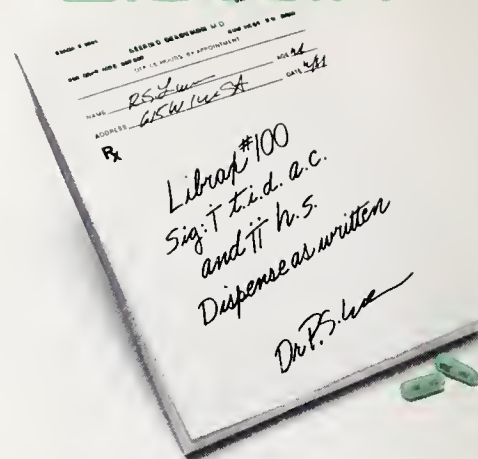
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INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.

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WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. *Drug Dependence:* Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy:* Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children:* Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular:* Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System:* Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal:* Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic:* Urticaria, rash, ecchymosis, erythema. *Endocrine:* Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System:* Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous:* A variety of miscellaneous adverse reactions have been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in the evening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in the morning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of January, 1980

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References: 1. Citations available on request from Merrell Dow Pharmaceuticals Inc., Cincinnati, Ohio 45215. 2. Hoekenga, M.T. et al: A comprehensive review of diethylpropion hydrochloride. In Central Mechanisms of Anorectic Drugs, S. Garattini and R. Samanin, Ed., New York: Raven Press, 1978, pp. 391-404.

Yankton District Medical Society (# 8)

W. F. Stanage, M.D.*

At the June 3, 1882, first annual meeting of the Dakota Medical Society in Milbank, Dr. S. B. McGlumphy of Yankton, was appointed a member of the Committee to draft the Constitution and By-laws. Four of the ten founding members, Drs. S. B. McGlumphy, D. F. Etter, and J. B. VanVelson of Yankton, and Dr. J. G. Conley of Elk Point were from what was to become the Eighth District. Dr. McGlumphy was elected the first permanent chairman and a delegate to the American Medical Association. Dr. Etter was elected censor. Soon afterwards, Dr. Camp of Springfield, Drs. Miller and Brecht of Yankton and Dr. Nutting of Marion Junction joined the Society.

The annual meeting of the Dakota Medical Society was held at Yankton, May 20 and 21, 1886. Twenty-five members were present. Tours were given through the Dakota Hospital for the Insane, the Government School for Indian Children, the Public School and Yankton College. It was reported that the members received "courteous entertainment" from the city of Yankton.

With the attainment of statehood, there was a change in the organization's name and the boundaries of the Society. The official name became the South Dakota State Medical Society. In 1903 the South Dakota State Medical Society underwent a reorganization of local societies. District VIII was composed of the counties of Union, Clay, Yankton, Bon Homme, part of Turner, Douglas, Charles Mix, part of Hutchinson, Gregory, Tripp, and Meyer.¹ In 1912 the Rosebud District was founded and Gregory and Tripp County became a part of that District.

The spring meeting of the District in 1925 was held at Sacred Heart Hospital. Dr. Melgaard of Sioux

City and Dr. Zimmerman of Sioux Falls conducted a "baby clinic."

In 1953 there were 41 members of the District, Dr. A. P. Reding was a councilor. In 1954 the members of the District Medical Society assisted in blood drawing for the American Red Cross Train. In December of 1956 Dr. J. A. Hoff was presented with a pin commemorating his 50 years of medical practice. An oral polio immunization program was sponsored by the District in September of 1962.

Meetings during 1968 were predominantly on the subject of the Medical School at the University of South Dakota. Dr. Knabe kept the Society informed as to the progress of the four year medical school. At the December 1973 meeting of the District, all the legislators from Bon Homme, Clay, Union, and Yankton counties were invited. A round table discussion was held regarding the Medical School.

South Dakota State Medical Association annual meetings in 1886, 1899, 1908, 1917, 1949, 1963, and 1966, were held in the District at Yankton. State presidents from District VIII have been; Drs. S. B. McGlumphy, J. B. VanVelson, F. Etter, C. C. Gross, L. C. Mead, G. S. Adams, S. M. Hoff, J. C. Ohlmacher, J. Patrick Steele, T. H. Sattler and D. B. Reaney.

In 1977 the Yankton District qualified for two councilors and in 1980, there were 56 members of District VIII.

Acknowledgment

Information provided by Sister Desideria of Sacred Heart Convent, Yankton, and the **History of the South Dakota State Medical Association, 1882 to 1956**, was most helpful in compiling this history.

REFERENCE

1. Dakota Medical Society Records, 1882-1904, pp. 182-83.

*Pediatrician, Yankton Clinic, 400 Park Ave., Yankton, SD.

Some Memories Of The Rosebud District Medical Society (# 10)

Robert Hayes, M.D.*

Unfortunately, the old secretary's book of the Rosebud Medical District has been misplaced. When I was secretary, I recall trying to keep it up-to-date. There had been a history from about 1939 to 1950. However, the first record was noted in 1903 and had been carefully and legibly inscribed by Dr. J. W. Cook of Fairfax, South Dakota. I met Dr. Cook in Bonesteel in 1951.

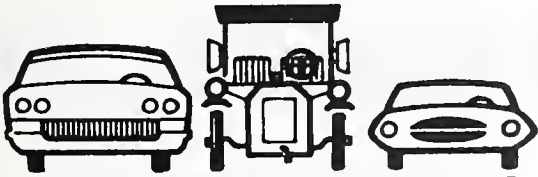
I recalled that at one time in 1916 (the book read) there were six practicing physicians in Dallas, South Dakota. The "Rosebud" was referred to as Gregory County and that territory which we now know as Tripp County, Todd County, and Mellette County.

*Previously in general practice in Winner, SD. Currently Chief, Division of Medical Services, Dept. of Health, Pierre, SD.

They listed 26 practicing physicians in that area. The same territory now is staffed by six physicians plus 3, 4, or 5 at the Rosebud Indian Hospital at Rosebud, SD (depending upon which month the information is gathered).

In the 1950's because we had so few physicians and district meetings were meaningless, we organized the "Rosebud Interprofessional Society" (RIPS) and included the local dentists, veterinarians, and pharmacists. We then could (and did) have scientific meetings, and a different discipline presented the scientific subject each meeting. We found that it did work and that we had someone to talk to other than ourselves.

Perhaps that model should be studied again in areas like the present Rosebud District (10th District Medical Society).



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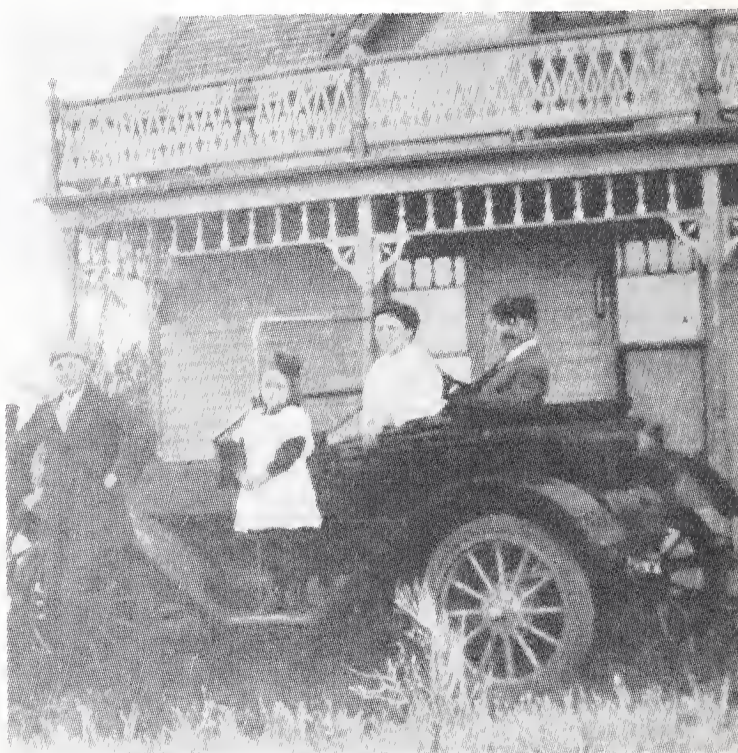
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Reprinted from *Doctors of the Old West* by Robert Karolevitz, Superior Publishing Co., Seattle, Washington, 1967, pg. 91. Books available through the author, Mission Hill, SD 57046.

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**for their outstanding and
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History of Northwest District Medical Association (#11)

Leonard M. Linde, M.D.*

In a previous publication, **The History of the South Dakota State Medical Association, 1882-1956**, prepared for the 75th anniversary of organized medicine in the state, it is recorded that in 1903 reorganization of the Medical Association districts created nine districts. The counties (or a portion of them) that now are included in the 11th District were then included in District 1. That was a rather large district with 14 1/2 counties listed as comprising that district. The first reference to a District 11 is made in a story quoting a physician from DeSmet from District 11. That city, of course, is not in the present District 11 and evidently sometime after 1929 reorganization produced the present District 11 with members practicing in Walworth, Potter, Dewey, Corson and Perkins counties.

Old files from this district contain some correspondence regarding requests for a charter for the 11th District. That correspondence is dated May and June of 1938. It appears then that the present 11th District was probably constituted in its present form in 1938.

This rural, sparsely populated district has one of the smallest memberships currently totaling about 13 members. In the recent history of District 11, it was an honor to have two State Association presidents come from this area. Dr. A. W. Spiry served as president in 1954-1955 and in 1977-1978 Dr. James Ryan filled that office.

For the past 10 years the members have met fairly regularly during the fall and winter months for monthly social, scientific and business meetings in Mobridge. Some physicians travel from 60 to 90

miles to attend. Wives (or husbands) have been invited to the social and scientific portion of the meeting. Members and their wives have found this form of combined meeting to be very successful. The present officers of the 11th District are; Dr. James Collins, Mobridge-President, Dr. Morris Benson, Lemmon-Vice President, Dr. Leonard Linde, Mobridge-Secretary and Dr. James Wunder, Mobridge-Councilor.

The members of this District feel that the observance of a Centennial Anniversary should bring to memory and to honor those pioneer physicians who served their communities unselfishly and often under very trying circumstances. A few of those departed physicians of this area that we feel need to be honored for their pioneer work are: Dr. G. E. Twinning, Dr. G. A. Sarchet, Dr. G. E. Lowe, all of Mobridge; Dr. W. A. George of Selby; Dr. C. E. Totten of Lemmon; Dr. T. H. Baer, Timber Lake; Dr. T. D. Jones, Bowdle; Dr. Frank H. Kramer of Dupree and Dr. L. C. Shockey of Pollock. Dr. Shockey was one of the true pioneer "horse and buggy" doctors. He began his practice in Mound City in 1897, later moved to Pollock where he practiced until retirement. He died at the age of 89 in 1960.

The district would also like to extend its appreciation to several physicians who are now retired or have left this area. These physicians also served their communities with devotion and gave of their time to support the Medical Association. We particularly remember our two past state presidents; Dr. A. W. Spiry and Dr. James Ryan both of Mobridge; also, Dr. B. P. Nolan of Mobridge, retired and living in Mobridge; and Dr. G. C. Torkildson of McLaughlin now in semi-retirement in Wisconsin.

*Family Physician, Mobridge, SD.

Whetstone Valley District Medical Society (# 12)

E. A. Johnson, M.D.*
Georgianna Bell**

The 12th District Medical Society known as the Whetstone Valley District Society received its charter from the South Dakota State Medical Association, August 1, 1923. This charter, which is the only remaining original charter of a district medical society of South Dakota to our knowledge, has been assigned to the State Medical Association office in Sioux Falls for safekeeping and display.

Of special historical importance to Milbank is the fact that the Dakota Medical Society was first organized as the Dakota Medical Society at Milbank, Dakota Territory in 1882. The original constitution and bylaws were adopted May 27, 1885.

Milbank was founded in 1880 and as the railroad moved west founding new towns, the medical profession migrated west also. The January, 1881 newspaper listed five physicians, one being a woman specializing in diseases of women and children; chronic cases a specialty. In May, 1881, Dr. H. G. C. Rose of Chicago, physician and obstetrician, came to hang his shingle. A week later, Dr. Rose was called to aid his fellow physician, Dr. Pine. The **Grant County Review** carried the following news item: "Dr. Pine was obliged to submit to a rather painful operation. He suddenly felt a pain in the body which he thought was a fish bone that he had swallowed. Dr. Rose was sent for, who discovered and very successfully removed a pin that had been firmly embedded crossways about 2 inches from the extremity. This was the first surgical operation performed by the new doctor."

The next meeting of the Society was scheduled to be held in Sioux Falls on June 12, 1890, at which time it was presumed the name would be changed to State Medical Society of South Dakota.

The 12th District Medical Society consists today of doctors from Milbank, Rosholt, Sisseton and

Webster. Other towns of Groton, Revillo, Waubay and Veblen had doctors in the past. Due to better transportation, it seems that only the county seats retained the doctors and population growth. The district physicians meet three times a year for dinner and medical subject review.

During the past thirty years, Milbank has had nine different doctors in practice. Dr. Lorin Drexell Dawson was with Dr. D. A. Gregory (who came in 1930) from 1947 to 1949. Dr. E. A. Johnson came in partnership with Dr. Gregory, December 14, 1950, and has remained in Milbank. Dr. Kurt Tauber came in 1946 and died August 2, 1951. Dr. Terrence McManus came in 1951 but left shortly to join Dr. Roscoe Dean at Wessington Springs and later became a psychiatrist in Sioux Falls, and later to Cherokee, Iowa. Dr. Rudolph Orgussar from Estonia was in Revillo from 1953 to 1954. Dr. Visvaldis Janavs from Latvia came to Milbank from Willow Lake to join Drs. Gregory and Johnson. Dr. Bernie Herzog joined Drs. Gregory, Janavs and Johnson in 1967 and two years later took a surgical residency at Yankton. He is located in Marshall, Minnesota.

Dr. Walter T. Judge came to Milbank in 1931 and practiced solo until his death in 1975. Three of his four sons became physicians, a grand tribute to their father.

On August 4, 1971, Dr. Ben Buentipo, Philippine Islands, came to practice in Milbank as a surgeon. In July 1980 he notified the community that he was leaving Milbank to join a clinic in Garland, Texas. He returned to Milbank in December until the facilities are completed in Texas sometime in the spring.

Dr. David A. Gregory retired in his eighties and following several years in the Whetstone Valley Nursing Home in Milbank transferred to the Masonic Home in Glasgow, Montana to be near his son, Dr. David Gregory, Jr. One of Dr. Gregory's oral histories included the following: One night during a bliz-

* Family Physician, Milbank, SD.

**Past SD State Auxiliary President, Webster, SD.

zard, the good doctor was called out in the country to deliver a baby. The doctor drove to within a half mile of the farm home, then with the snow impassable with the car, trudged through the snow the remaining distance. After delivering the baby, he asked the husband why hadn't he come out to meet him with the horse and sled knowing full well that the car couldn't make it. "Why doc" the husband said, "I'd never take my horses out on a night like this!"

In the past thirty years, seventeen young men and four young women from the Milbank area have entered medical school and two of them may soon return to the area to establish family practice.

Rosholt is a small town in the very northeast corner of South Dakota. It is the first South Dakota community west of the Minnesota border and south of the North Dakota border. The population of 500 has remained relatively stable over the past 25 years.

A Dr. Allen practiced in Rosholt until 1945 when he died, leaving the community without a physician for fifteen years. In 1959, the town board decided to begin a physician recruitment program and send representatives to Winnipeg, Canada, to conduct a search.

At that time, Dr. Joseph Kass, who had emigrated from Hungary, was interning in Winnipeg. He was invited to visit Rosholt. He, his wife, Kathy, and six year old son Tommy were favorably impressed with the fine community, the hunting and fishing and the lovely surroundings. Dr. Kass agreed to move to Rosholt to practice when a clinic became available. Plans for the new clinic were made, money was donated by local citizens, and the clinic building was completed in 1960. Dr. Kass began his practice in the autumn of that year and eventually purchased the clinic.

He is on the medical-surgical staff of the Wheaton, Minnesota Hospital near Rosholt as well as the Coteau Des Prairie Hospital in Sisseton and the Day County Hospital in Webster. Since Dr. Kass has been in Rosholt, a fifty bed nursing home has been built there. This enables the elderly to be cared for near their homes and families.

Mrs. Kass, formerly a soprano with a Hungarian opera company was recently the subject of a feature story in the periodical, **M.D.'s Wife**.

In 1956, Sisseton had two clinics and three hospitals. The Public Health Service Hospital of about 20 beds was served by Dr. Harry Brauer and Dr. W. C. Brinkman on a contract basis. They maintained their private practices at the Sisseton Clinic and were on the staff of the Community Hospital. That hospital was located on the first floor of an apart-

ment building and contained about twenty beds. The Tekakwitha Hospital, associated with Tekakwitha Clinic, was owned by Dr. Percy Peabody, who practiced there. It eventually became part of the Tekakwitha Nursing Home which was recently razed. The Community Hospital and the Tekakwitha Hospital were replaced in 1967 by the new, modern Coteau Des Prairie Hospital.

Dr. Ed Batt, fulfilling his military obligation through the Public Health Service, was assigned to the Public Health Service Hospital in Sisseton in September, 1956. He was the first Public Health Service physician to serve that hospital for several years since the Public Health Service had recently taken responsibility for Indian health from the B.I.A. Upon completion of Dr. Batt's Public Service obligation in 1958, he joined Dr. Brinkman and Dr. Brauer in practice.

Dr. Percy Peabody passed away in 1965. Dr. Brinkman retired in 1970 and has remained in Sisseton. Dr. Brauer moved to Mankato, Minnesota in 1970 and continues to live there. During the early 1970's, Dr. Steve Sullivan and Dr. Kewal Verma practiced in Sisseton for a short period.

In 1972, Dr. Tom Ward, a National Health Service Corps physician arrived to serve both Sisseton and Veblen. At about that time the Sisseton Clinic Building closed and a new office building was constructed. This building connected to the new hospital and also contained office space for one dentist. It was renamed Coteau Des Prairie Clinic in 1976.

Dr. David Oey and Dr. Glen Oey arrived to practice medicine in Sisseton in 1975. Dr. David Oey continues to practice there. Dr. Glen Oey moved his practice to Watertown in 1978. In 1976, Dr. Batt joined the faculty of the University of South Dakota School of Medicine and moved to Sioux Falls where he passed away in 1978. Dr. David Staub, a National Health Service Corps physician, arrived in 1976 as did Dr. Valentin Mendoza. Both continue in private practice. Dr. Joseph Kass of Rosholt has provided surgical services for the Sisseton area since 1960.

The year 1956 saw the dawning of a new era for medicine in Webster, a community with a proud medical history. In November of 1955, the voters of that community decided, by a margin of 2767 to 795, to replace the old Peabody Hospital (which had housed a school of nursing and had been the birthplace of many of those voters) with a new, modern hospital. At the time of that vote, the staff of the Peabody Clinic and Hospital was recorded as follows: Dr. Faris Pfister, Dr. William Duncan, Dr. Walter H. Karlins, Dr. Joseph Lovering, Dr. Dagfin Lie, and Dr. L. W. Keller. In addition, there was one medical technologist, one x-ray technologist, 19

nurses, 18 nurses aids and 14 other employees. The new 40 bed hospital was completed in 1958, one year after the new 58-bed Bethesda Nursing Home was opened. Dr. Faris Pfister passed away suddenly in 1957 before these new facilities were available.

Dr. Loren Amundson joined the group in 1959 and remained for four years, moving his practice to Sioux Falls in 1963. Dr. Walter Kitzler arrived in 1965 and remained for one year. During that year, Dr. Duncan died. In January of 1965, the new clinic building, financed by the Webster Business and Professional Association, was opened. It was named Day County Medical Center and provided office space for four physicians and two dentists as well as containing reception rooms, business offices, x-ray and laboratory areas.

In 1968, Dr. Karlins retired due to illness. He passed away in 1970. In 1969, two of the three remaining physicians moved away, Dr. Lie to join the staff of the Veteran's Administration Hospital in Sioux Falls and Dr. Keller, the Veteran's Administration Hospital in Middleton, Wisconsin. In April of that year, the county purchased the clinic building. At about that time, seeing a crisis in health care availability developing for the Day County area, the Webster Development Corporation assumed a major responsibility for physician recruitment.

First, a committee analyzed the problems to be solved in order to recruit physicians for Webster and devised a plan for solving them. Housing was seen as a major hurdle. The Development Corporation answered this problem by building one modern house and purchasing another for the use of new physicians. It was proposed that the houses be rented to the physicians at a rate sufficient to repay the mortgage with the rental to apply toward purchase if desired.

A second major problem seen by small town physicians and their families is the lack of leisure time. The solution proposed was a strictly enforced call system in which each physician in turn was totally responsible for all emergencies (including obstetrics) in a 24-hour weekday period. Weekends were to be rotated as a Saturday-Sunday unit. All physicians were to have unlisted telephone numbers, all emergency calls were to be handled through the hospital switchboard and only the physician on call was to be called. This system guaranteed 24-hour a day emergency room coverage, a status lacking in many larger hospitals at that time.

A third hurdle, that of moving costs and initial living and office expenses was answered by the Development Corporation underwriting moving expenses and guaranteeing a minimum income during the first year of practice.

The fourth problem, that of selling the commu-

nity to the prospective physician and his (her) spouse was solved by providing them with an expense free trip to Webster which included tours of the area, parties which local citizens attended and an opportunity to observe all aspects of the practice and of life in the area.

The only remaining problem was finding the physicians to recruit. This was done through advertisements in journals, visits to medical schools and teaching hospitals, and investigation of word of mouth reports about available physicians.

The results were successful and in 1969, four new physicians arrived to practice in Webster. They were: Dr. Lloyd C. Vogelgesang, family practitioner; Dr. Ben Buentipo, surgeon; Dr. Richard Friess, family practitioner; and Dr. Eldon Bell, preventive medicine specialist. They joined Dr. Joseph Lovering, surgeon, who remained on the staff. Dr. Bell assumed Dr. Lie's position as county coroner enabling Day County to remain one of the few counties in the state with a physician serving in that position.

In 1970, Dr. Friess moved to Sioux Falls where he continues to practice and Dr. Buentipo moved to Mora, Minnesota. In 1971, Dr. Lawrence F. Nelson, a family practitioner joined the staff and remains there today as do Drs. Vogelgesang and Bell.

In 1976, Dr. Arnath Unahalekhaka, surgeon, joined the staff. He moved his practice in 1978 to Copper Mountain, Tennessee. In the autumn of 1978, a contract was signed with Hospital Affiliates International for administration of the hospital. Dr. Lovering passed away during the same autumn after a short illness.

In 1979, a major renovation and addition to the Day County Hospital was completed. This addition increased space in the Nurses Station, modernized the nursery and intensive care unit and improved efficiency in other areas of the hospital while retaining the same patient capacity. This addition was dedicated by Gov. William Janklow who was introduced by former Gov. Sigurd Anderson of Webster.

Also in 1979, Dr. Stanley Gerrick arrived to practice surgery and remained for one year.

Presently, an addition to the Day County Medical Center is under construction which will provide office space for two additional physicians. This will be opened in the spring of 1981 and recruitment of physicians for those new offices is in progress.



SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS
3001 South Holly Avenue
Sioux Falls, SD 57105



Facts About the Specialty of Family Practice

The specialty of family practice was officially recognized February 8, 1969, when the American Board of Medical Specialties and the Council on Medical Education of the American Medical Association approved a primary certifying board in the specialty. The application for a certifying board was submitted by the then American Academy of General Practice (now the American Academy of Family Physicians) and the Section on General Practice of the AMA. The application for establishment of a certifying board incorporated a set of "Essentials for Residency Training in Family Practice" approved by the AMA's Council on Medical Education and House of Delegates and the Academy's Congress of Delegates.

The American Board of Family Practice (ABFP) is a free-standing organization whose purpose, as with all other medical specialty boards, is to conduct examinations to measure competence in a special field of medicine (family practice) and to grant certification on the basis of performance in these examinations. Its membership consists solely of its officers and board of directors. Physicians whom it certifies are called diplomates. The structural organization of the new ABFP took shape June 22, 1969, following approval the previous February of a primary certifying board in family practice by the American Board of Medical Specialties.

The American Board of Family Practice conducts examinations and grants certification to family physicians who meet its qualifications and pass the examination. Ten examinations have been held. More than 22,000 diplomates have been certified.

The ABFP also requires recertification by examination every six years, the only certifying board to do so. The first recertification examination was held in the fall of 1976, with some 1,400 diplomates of the 1970 class taking the examination. The ABFP is independent of the Academy. AAFP members do not have to take the board examination to remain Academy members, nor is board certification a requirement for AAFP membership.

The specialist in family practice is an examination-certified family physician who:

- (1) Serves the public as the physician of first contact and means of entry into the health care system;
- (2) Evaluates his patients' total health needs, provides personal medical care within one or more fields of medicine, and refers patients when indicated to appropriate sources of care while preserving the continuity of his own care;

- (3) Assumes responsibility for his patients' comprehensive and continuous health care and acts as a coordinator of his patients' health services, and
- (4) Accepts responsibility for his patients' health care, including the use of consultants, within the context of their environment—the family or comparable social units and the community.

Vice President Candidate

Mike Brown, M.D., Spearfish, has been nominated by the Board of Directors for a three year term as Vice President, SDAFP. The election will be held during the annual business meeting at the Black Hills Summer Seminar, August 14, 1981, in Rapid City.

Other candidates nominated include Vice President Herb Soloum, M.D., Tyndall, for President-Elect and L. H. Amundson, M.D., Sioux Falls, current Secretary-Treasurer, for another term in this office.

Ray Gene Nemer, M.D., Gregory, will become President at the August meeting and Larry Finney, M.D., of Sioux Falls and Chuck Swanson, M.D. of Pierre continue as Vice Presidents.

President Bill Tschetter, M.D., Rapid City, will replace Buron Lindbloom, M.D., of Pierre, on the Board as Past President.

Plan Now To Attend

The AAFP's Annual Scientific Assembly, September 21-24, 1981, with special events for spouses. This event will be held in Las Vegas, Nevada.

NEW BYLAWS

SDAFP members attending the Annual Business Meeting in Rapid City, August 14, 1981, will be asked to vote on a revised set of Bylaws. This will conform to changes made by AAFP, deleting the Constitution and incorporating it into uniform Bylaws suggested for constituent (state) chapters. A copy of the proposed Bylaws is on file at the state office for members, upon request.

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President's Page



What's Ahead?

Events of the future are now more than ever influenced by economic developments. In the past few years, the Medical Association has found strength to resist administrative proliferation and third party intrusion by reacting in unity and by planning ahead. Many concessions and delegations of responsibility have been made in the years since Medicare began. Most of these were popularized by "the doctor shortage" approach and were forgiven by the medical industry for the sake of governmental cost accounting. The pressure has been kept on the medical profession by liberal politics with a media to match.

In the years ahead, we face decreasing federal health budgets and greater competition for health allocated dollars. With this will come first, cuts in compensation to physicians, some form of rationed services, primary or secondary, and last of all will come a decrease in the administrative costs of federal programs. Increasing roles by the state government were developed in administering health allocated dollars. It is very likely that big government will attempt to control costs by increasing regulation, which never has worked but which is politically expedient and comfortable for bureaucratic power-base preservation. Paralleling these developments and the increase in numbers of physicians, a new era of medical economics will be seen. Increasing loss of self-determination dictated by economics can well be the outcome for our physicians. Some will be voluntarily in pre-paid plans and third party negotiation fee plans. Hospital employed practice and increases in government employed physicians are

likely. Internal medical politics will be similarly affected by an influx of people not committed to free enterprise.

Whereas these impressions may sound pessimistic, they are probably a true possibility. What can be done? A high level of intensity must be maintained on the commitment to withdraw federal and state regulations as budgetary decreases develop. It would be easy to lose the battle here. Physicians must remain unified if they are to succeed to continue as a free enterprise entity.

To manage the changes and developments which lie ahead, we must rely upon the responsible attitudes and actions taken by the South Dakota State Medical Association as expressed by the participating members.

What lies ahead for your President? He sees himself with the Dr. Jeckel and Dr. Greed column in the *SOUTH DAKOTA JOURNAL OF MEDICINE*, for your thought and entertainment. Thank you for allowing me to serve as your President of the Medical Association. I enjoyed it.

Very sincerely yours,

Winston B. Odland, M.D., President
South Dakota State Medical Association

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A Brief History Of South Dakota Academy Of Ophthalmology & Otorhinolaryngology

Stanley B. Altman, M.D.*

On the 22nd of March 1931, a meeting was held at the request of Dr. H. C. Peabody, president of the South Dakota State Medical Society. The meeting was held in Huron, SD and in attendance were Doctors J. D. Alway, I. Miller, A. Johnson, C. E. Robbins, L. N. Grosvenor, Cook, and H. L. Saylor. The purpose of the meeting was to consider the prospect of forming a South Dakota Academy of Ophthalmology & Otolaryngology to be in some way connected with the State Medical Society. Dr. Grosvenor was elected temporary president and Dr. Saylor was elected temporary secretary. The next meeting would be held in connection with, and at the time of the next meeting of the State Medical Society, which was on June 2, 1931, in Aberdeen, SD. Present were Doctors Grosvenor, Miller, Peabody, Alway, Johnson, Gregg, Kelly, and Saylor from South Dakota; along with North Dakota Academy members, Doctors Wicks, McCannell, Obsthahl, and Coustaus. At this second organizational meeting Dr. Grosvenor was elected president, Dr. Gregg vice president, and Dr. Saylor secretary-treasurer. These officers-elect were to constitute a committee to create a constitution and bylaws for the newly formed South Dakota Academy of Ophthalmology & Otorhinolaryngology.

The early years of the Academy meetings were held semiannually. The midyear meeting taking place usually just prior to Christmas, and the regular meeting was held in conjunction with the meeting of the South Dakota State Medical Society. During these early years the main activity of the Academy was educational. The programs were varied and consisted of presentations by members of the South

Dakota Academy. After each particular paper or presentation a discussion was held either by a panel or by the membership as a whole. Often times guest speakers were brought in for scientific presentations. These guest speakers were prominent men from universities or private practice from all parts of the country. Most were from either the University of Minnesota or the Mayo Clinic, however speakers from as far away as Boston, Denver, Iowa City, and Chicago were guests of the Academy.

The past twenty years or so the practice and indeed the whole character of medicine has been changing. This is true also of ophthalmology and otolaryngology and therefore the function of the Academy. Dissemination of scientific information is still the prime goal of the Academy. However more and more, the Academy has been forced to address itself to non scientific topics such as governmental interference, relationships between medical and non-medical personnel, hearing aid distribution and regulations, and most recently optometry and optometric use of prescription pharmaceutical agents. Members of the Academy have been active in politics, attending legislative sessions in Pierre lobbying for and against certain bills.

The Academy's financial picture has also changed with the times. Originally the dues were \$2.00 per year and many years the dues were omitted because there were sufficient funds in the treasury. One of the reasons for this may be that lunch for ten after the 1934 meeting cost \$5.05. The South Dakota Academy of Ophthalmology & Otorhinolaryngology continues to provide its membership with continuing education and a forum for legislative action. Through its members, the people of South Dakota have available the best medical care for eye, ear, nose, and throat.

*Ophthalmologist, Aberdeen, SD.

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The Roots Of Family Practice In South Dakota

L. H. Amundson, M.D.*

Preface

"I can honestly say that I have very few complaints or I wouldn't have stuck out 12 years of practice here. The compensating factors have been too great to lure me to a higher fee area, one with greater cultural attractions or scenic beauty or better climate. The biggest factor in my satisfaction is our medical practice group (which now includes a 4th doctor in our practice). Group practice has eliminated the problems of vacations, time off, and attendance at things out of town or out of state. It also has enabled us to broaden the scope of services and abilities we are able to offer our patients. It also has cut down our individual investment in first rate equipment, buildings and facilities, enabling us to enjoy the above things and yet make a good income at modest cost to our patients who for the most part are hard pressed financially. We also have the satisfaction of knowing that our people are being covered 24 hours a day every day of the year with minimal sacrifice on our part."

From **Observations and Suggestions From Rural Doctors of South Dakota**, solicited and compiled by Roscoe E. Dean, M.D. of Wessington Springs, South Dakota circa 1970.

Many Americans who lived during the Forties and Fifties probably look back on those decades with nostalgia—but not the general practitioners/family doctors who practiced then. For these physicians, the "good old days" of those decades were times of frustration and discouragement.

Although they enjoyed the esteem of their patients and rightly believed the medical care they provided to be of great importance, they found their numbers dwindling and their status as physicians in

question. At that time, only one of every ten medical students planned to enter general practice. The GP was the low man on the medical totem pole and many doctors and medical educators were sure that he was on his way out. Just as distressing, many general practitioners were denied the privilege of admitting their patients into hospitals and continuing their supervision there.

Background

The reasons for the GP's dilemma were complex and intricately linked to the major events happening on the medical scene and in the nation:

First, the rapid scientific and clinical advances of that era had given rise to the belief that no physician could be schooled adequately in all areas of medicine. Hence, the proper approach seemed to be specialization so that a physician could concentrate on learning everything about one particular area or organ rather than trying instead for a broad, basic knowledge of the entire person. A natural follow-up was the assumption made by most medical students that to be a general practitioner was somehow to be the lesser doctor and to practice "second-rate" medicine.

Second, World War II and its aftermath greatly accelerated the trend toward specialization. During the war, board certified physicians automatically received the higher rank, more status and more pay. After the war, subsidies for graduate medical education under the G.I. bill made it possible for physicians to live comfortably while doing their residency work and at the same time gave hospitals the house staffs they needed.

These factors worked to the mutual advantage of hospitals and physicians interested in specializing. The only one not taken into account was the general practitioner. Where could he turn to continue his graduate medical education?

*Secretary-Treasurer, South Dakota Academy of Family Physicians; Associate Director, Sioux Falls Family Practice Residency Program; Professor of Family Medicine, University of South Dakota School of Medicine.

The answer was simple—nowhere. Until 1950 there were no graduate programs available in general practice. Even after these programs were set up they gained little ground. The idea of “specialty” training for a “generalist” seemed incongruous to the medical community. The residents themselves lacked the identity and status accorded other graduate trainees and the programs faltered.

In the early 1960s, four major studies sought the answer to the question, “What’s wrong with the delivery of health care in America?”:

- 1) The National Commission on Community Health Services, under the chairmanship of Marion B. Folsom, former Secretary of Health, Education and Welfare.
- 2) The Citizen’s Commission on Graduate Medical Education, headed by Dr. John Millis, President of Western Reserve University.
- 3) The Ad Hoc Committee on Education for Family Practice, established by the Council on Medical Education of the AMA, under the chairmanship of Dr. William R. Willard, then Dean of the University of Kentucky Medical School.
- 4) The Conferences of the Family Health Foundation of America, chaired by Dr. Ward Darley, previously the President of the University of Colorado and Executive Director of the Association of American Medical Colleges.

These four studies, independent of each other, arrived at a single conclusion: “Every individual should have a personal physician who is the central point for integration and continuity of all medical and medically related services to his patient.”

American Academy Founded

It is against this background that the American Academy of General Practice (the name was changed to the American Academy of Family Physicians in 1972) was founded in 1947. As one editorial of that time said, the GP had realized at last “that the day when organized medicine was going to do something for him had been too long delayed and there was barely time remaining for him to do something for himself.”

The Academy was created to serve as the national spokesman for the nation’s general practitioners and as a unifying force among them. It was welcomed by them almost without reservation—witness the fact that membership sky-rocketed from zero to 2,000 in the first six months alone. Its objectives were representation for the general practitioner, opportunity for postgraduate study in his field and recognition of his scientific attainments.

The last point, recognition of the generalist’s sci-

entific attainments, was the first task to which the Academy directed itself, working for many years in conjunction with other medical organizations to secure fair access to hospital privileges for its members. This battle has been won, in part, because Academy members showed themselves to be as much—or more—interested in continuing their education than their “specialist” colleagues.

The most distinctive element about the Academy has always been its emphasis on continuing education. Each AAFP member is required to complete 150 hours of continuing medical education every three years in order to retain membership. This requirement was unique among national medical organizations until the 1970s. It served as the model for the AMA’s voluntary Physicians Recognition Award. The fact that it was a part of the original AAGP Bylaws shows the intense interest Academy founders had in assuring quality medical care from its members.

South Dakota Academy Follows

The formation of a South Dakota Academy of General Practice was first discussed at a meeting held at McKennan Hospital Library in Sioux Falls on June 4, 1950, called by A. P. Reding of Marion, South Dakota. Physicians present were: A. P. Reding; M. Drobinsky of Estelline; L. L. Parke of Canton; and L. J. Pankow, H. O. Kittelson, and J. A. Kittelson of Sioux Falls.

The charter date was later set as January 1, 1951, and the first organizational meeting was held at the Marvin Hughitt Hotel in Huron on January 13, 1951. Eleven members had paid initial dues and twenty members attended this first meeting.

Early support of medical education was evidenced by passage of a resolution at the first meeting. This resolution requested additional legislative support of \$100,000 to finish and complete the new Andrew Lee Medical School Building being built at USD.

AAGP Executive Director Mac F. Cahal attended the June 6, 1951 chapter meeting in Aberdeen. He cautioned the twenty-five members present that hospitals prefer to be staffed with “specialists” because of their intern teaching. He used this as an example of the fact that hospitals “are trying to push themselves into the practice of medicine and the control of medical practice.”

Evidence of chapter concern for “postgraduate” medical education opportunities for its members was resolved early in the life of the chapter. The first scientific meeting was held April 11-12, 1953 in Huron with 48 members attending. Seventy-five members attended the 1954 scientific meeting where Dr. Phillip Thorek of Chicago was one of five out of

state speakers presenting papers on "Accident Medicine." Scientific and business meetings of SDAGP were usually held in Huron during those first years due to being "centrally located."

The first Memorial Scholarship was established in 1954 in memory of Dr. John A. Kittelson of Sioux Falls, the first Secretary-Treasurer of SDAGP.

AAFP Growth

"Growth" has been the keynote of the Academy since its founding. Space requirements for the headquarters offices have outgrown three buildings and the staff has increased from three to more than 120. Current membership stands at over 50,000 members, making it the nation's largest medical specialty society and second largest medical association.

Largely due to Academy efforts, family practice was designated as medicine's twentieth specialty in 1969, a recognition by the medical community of the fact that a physician can be a specialist in breadth as well as depth. By 1980, family practice programs were established in more than 85% of the nation's 134 medical schools, and 382 family practice residencies in medical schools and community hospitals have graduated over 8,500 family doctors and are now training 6,351 FP residents. For this to have occurred in so few years led one science writer to term family practice "potentially the most far-reaching reform in medical education in our time."

Here At Home

South Dakota Academy membership now exceeds 250, made up of 160 active, affiliate, and life members; all South Dakota Family Practice Residents as resident affiliates; and one-half of USDSM third and fourth year students as student affiliates. Over 100 active members are ABFP Diplomates.

In addition to growth, evidence of stability and continuity exists in the South Dakota chapter. Drs. Art Reding of Marion and H. O. Kittelson of Sioux Falls who attended the pre-formative meeting of our Academy still practice in their respective cities. Dr. Reding was cited by the South Dakota Academy for nineteen years as a delegate to the Congress of Delegates, AAFP during a dinner held in his honor at the 1973 national meeting in Denver.

Other SDAFP members have also made significant contributions to the chapter and to family practice education in South Dakota. Drs. Paul Aspaas of Dell Rapids and L. J. Sweeney of Sioux Falls were instrumental in re-developing the Black Hills Summer Seminar into its current format in 1970. Drs. Gary Welsh of Lead (now practicing in Rapid City), and Mike Brown of Spearfish envisioned and developed the first Black Hills Winter Ski Seminar in

1976.

Dr. L. J. Sweeney left his Sioux Falls private practice in 1973 to start the Sioux Falls Family Practice Residency, serving as Director until his move to Sun City, Arizona in 1979. Dr. Dick Friess of Sioux Falls, working with the chapter legislative committee, spearheaded the first successful state funding bill for family practice residency support in 1976. Dr. L. H. Amundson of Sioux Falls left private practice in 1974 to become the founding chairman of the Department of Community and Family Medicine for the new four year medical school, serving as Chairman until 1979. Both the residency and the department have been housed in Sioux Falls since their beginnings.

Profiles of practice of South Dakota Academy members have been chronicled in the SD Chapter News, the result of AAFP surveys. Likewise, practice profiles of the Family Practice Residency have been the subject of clinical research projects and publications in referred journals.

The South Dakota Academy utilizes resources of the SDSMA for secretarial services, seminar planning, and staff time during seminars. This interaction has allowed a closer communication between our two organizations, to the mutual benefit of both.

Some Frequent Questions

Does the Academy duplicate the activities of any other medical organization?

No—not the American Medical Association, the specialty boards, nor any other national medical group. While it willingly cooperates with other agencies whenever appropriate and desirable, its programs are solely and specifically geared toward ensuring that all family physicians have the right to practice the type of continuing, comprehensive, quality medicine for which they are trained.

What is the relationship between the Academy and the AMA?

The Academy recognizes the American Medical Association as the parent organization of the medical profession in America. Its aim is to work in cooperation and harmony with the AMA while maintaining its sovereignty as the voice of family physicians.

The Academy's Executive Committee and the delegate and alternate delegate to the AMA House of Delegates serve as the AMA Section Council on Family and General Practice. The AAFP Congress of Delegates elects one delegate and one alternate delegate to the AMA House of Delegates.

In addition, there is joint AMA and Academy representation on several other councils and committees.

What is the Academy role regarding family practice in hospitals?

The Academy maintains that every family physician should have equal opportunity with other specialists to qualify for hospital privileges but believes privileges should be extended only on the basis of competency.

The Academy believes that integration of competent family physicians into hospital medical staffs is in the best interests of both the public and the profession.

To achieve this goal, the Academy maintains close contact with hospital staff activities to ensure that family physician representation is preserved and strengthened. The commission also cooperates with other groups such as the American Hospital Association, the AMA and the Joint Commission on Accreditation of Hospitals to expand hospital services and raise standards.

What is the Academy’s relationship to the American Board of Family Practice?

The Academy and the AMA Section on Family and General Practice were joint applicants and sponsoring organizations for the establishment of the American Board of Family Practice. When the Advisory Board for Medical Specialties and the AMA Council on Medical Education approved the creation of this recognized certifying board in February 1969, the ABFP was established as an independent, self-governing corporate entity. Under the ABFP bylaws, 10 of the 15 members of the ABFP Board of Directors must be members of the Academy.

Family Practice Thrives . . .

In short, family practice has ushered in the “good new days” for family doctors. Added emphasis has been given to their place in the medical spectrum and the kind of family-centered, “people” medicine they offer is in demand.

One sector of the nation that has needed no convincing of the specialty’s worth is the public. People today know that having a family doctor is a comfort, a relief, sometimes a rarity—but always a luxury because of the peace of mind it brings. Because the specialty will make more family doctors available, the public’s acceptance of it has been whole-hearted and instantaneous.

They may not care whether the doctor they see is termed a “family practice specialist” or a “general practitioner” or even care about the difference between the two, although there are important differences. Yesterday’s GP, for instance, often provided only episodic care and had little training in preventive medicine or helping him recognize the psychological aspects of illness. The specialist in family practice—the family doctor—is being trained to provide continuing, comprehensive care within the context of the family unit and receives special background in the behavioral sciences. Preventive medicine will be these physicians’ forte. In short, they are being **taught** during medical training what general practitioners had to learn by years of experience.

This form of medical practice, based on the solid foundation of classic general practice, is the primary link between the public and an increasingly impersonal “health care system.” It is the particular medical specialty for the people and the discipline of the “family doctor” of the future.

But the public’s major concern is simply that this kind of doctor be available—to talk to, to cure a sore throat, to handle an emergency.

Congratulations!

The South Dakota Academy of Family Physicians congratulates the South Dakota State Medical Association on 100 years of service to Dakota Territory. Thanks for your help. Together we can do better.

CHAPTER OFFICERS			
Year	President	Delegates	Secretary-Treasurer
1951-52	F. F. Pfister, Webster	L. J. Pankow, S.F. J. A. Kittelson, S.F.	J. A. Kittelson, Sioux Falls
1952-53	A. P. Reding, Marion	A. P. Peeke, Volga J. C. Hagin, Miller	J. A. Kittelson, Sioux Falls
1953-54	R. A. Buchanan, Huron	M. Drobinsky, Estelline J. C. Hagin, Miller	J. A. Kittelson, Sioux Falls
1954-55	J. C. Hagin, Miller	A. P. Reding, Marion J. C. Hagin, Miller	Magni Davidson, Brookings
1955-56	H. J. Grau, Rapid City	A. P. Reding, Marion J. C. Hagin, Miller	Magni Davidson, Brookings
1956-57	H. R. Wold, Madison	A. P. Reding, Marion J. C. Hagin, Miller	Magni Davidson, Brookings
1957-58	C. A. Johnson, Lemmon	A. P. Reding, Marion J. C. Hagin, Miller	Magni Davidson, Brookings

1958-59	C. J. McDonald, Sioux Falls	A. P. Reding, Marion J. C. Hagin, Miller	H. R. Wold, Madison
1959-60	G. J. Bloemendaal, Ipswich	A. P. Reding, Marion J. C. Hagin, Miller	H. R. Wold, Madison
1960-61	Magni Davidson, Brookings	A. P. Reding, Marion J. C. Hagin, Miller	H. R. Wold, Madison
1961-62	Preston Brogdon, Mitchell	A. P. Reding, Marion J. C. Hagin, Miller	H. R. Wold, Madison
1962-63	Preston Brogdon, Mitchell	A. P. Reding, Marion J. C. Hagin, Miller	H. R. Wold, Madison
1963-64	C. F. Binder, Chamberlain	A. P. Reding, Marion J. C. Hagin, Miller	H. R. Wold, Madison
1964-65	C. F. Binder, Chamberlain	A. P. Reding, Marion J. C. Hagin, Miller	H. R. Wold, Madison
1965-66	H. R. Wold, Madison	A. P. Reding, Marion J. C. Hagin, Miller	W. O. Hanson, Huron
1966-67	G. R. Bell, De Smet	A. P. Reding, Marion E. T. Lietzke, Beresford	W. O. Hanson, Huron
1967-68	P. K. Aspaas, Dell Rapids	A. P. Reding, Marion E. T. Lietzke, Beresford	W. O. Hanson, Huron
1968-69	J. S. Devick, Colton	A. P. Reding, Marion E. T. Lietzke, Beresford	P. K. Aspaas, Dell Rapids
1969-70	D. L. Scheller, Arlington	A. P. Reding, Marion E. T. Lietzke, Beresford	P. K. Aspaas, Dell Rapids
1970-71	D. L. Scheller, Arlington	A. P. Reding, Marion E. T. Lietzke, Beresford	P. K. Aspaas, Dell Rapids
1971-72	L. J. Sweeney, Sioux Falls	A. P. Reding, Marion P. K. Aspaas, Dell Rapids	L. H. Amundson, Sioux Falls
1972-73	L. J. Sweeney, Sioux Falls	A. P. Reding, Marion L. H. Amundson, Sioux Falls	L. H. Amundson, Sioux Falls
1973-74	B. C. Lushbough, Brookings	L. J. Sweeney, Sioux Falls L. H. Amundson, Sioux Falls	L. H. Amundson, Sioux Falls
1974-75	W. J. Kovarik, Rapid City	W. J. Kovarik, Rapid City L. H. Amundson, Sioux Falls	L. H. Amundson, Sioux Falls
1975-76	R. W. Friess, Sioux Falls	R. W. Friess, Sioux Falls L. H. Amundson, Sioux Falls	L. H. Amundson, Sioux Falls
1976-77	R. W. Friess, Sioux Falls	R. W. Friess, Sioux Falls L. H. Amundson, Sioux Falls	L. H. Amundson, Sioux Falls
1977-78	B. O. Lindbloom, Pierre	R. W. Friess, Sioux Falls L. H. Amundson, Sioux Falls	L. H. Amundson, Sioux Falls
1978-79	B. O. Lindbloom, Pierre	R. W. Friess, Sioux Falls L. H. Amundson, Sioux Falls	L. H. Amundson, Sioux Falls
1979-80	James E. Ryan, Mobridge	R. W. Friess, Sioux Falls L. H. Amundson, Sioux Falls	L. H. Amundson, Sioux Falls
1980-81	William R. Tschetter, Rapid City	R. W. Friess, Sioux Falls L. H. Amundson, Sioux Falls	L. H. Amundson, Sioux Falls

1980-81 Officers, Delegates, Alternate Delegates:

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 President Elect Raymond G. Nemer, Gregory
 Vice-President Herbert A. Saloum, Tyndall
 Vice-President Lawrence W. Finney, Sioux Falls
 Vice-President Charles Swanson, Pierre
 Past President Buron O. Lindbloom, Pierre
 Secretary-Treasurer L. H. Amundson, Sioux Falls
 Delegates: L. H. Amundson, Sioux Falls
 Richard W. Friess, Sioux Falls
 Alternate Delegates: Buron O. Lindbloom, Pierre
 William R. Tschetter, Rapid City

because they take satisfaction from being able to serve in an area where they are truly needed.

- Most of the doctors are concerned about the future of the rural hospital.*
- The universal plea is for more help which means more doctors.*
- Apparently the majority of doctors answering feel the medical needs of rural South Dakota can best be served by strategically located 5-8 man groups.*
- How to get these groups started is a challenge."*

Postscript

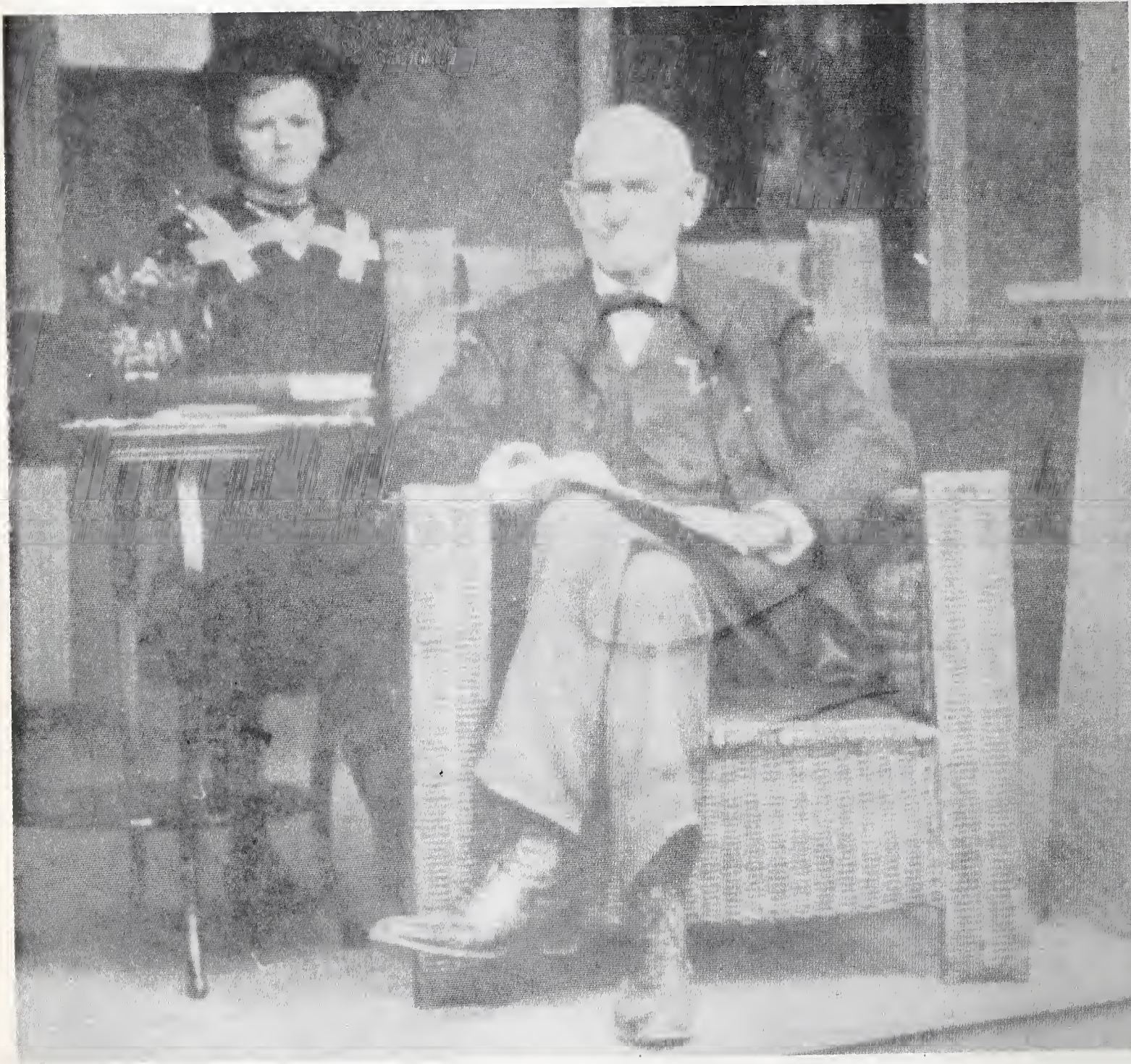
"To summarize:

- The majority of doctors serving in small communities are there because they want to be, and*

From **Observations and Suggestions From Rural Doctors of South Dakota**, solicited and compiled by Roscoe E. Dean, M.D. of Wessington Springs, South Dakota circa 1970.

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When President Abraham Lincoln needed a governor appointee for the Dakota Territory, he turned to his family physician, Dr. William Jayne of Springfield, Illinois. Then in his mid-thirties, Doctor Jayne traveled to Yankton, D.T., where he established the government in a log house. He was later elected territorial delegate to congress, after which he returned to Springfield where he was politically, financially and professionally successful.

Reprinted from *Doctors of the Old West* by Robert Karolevitz, Superior Publishing Co., Seattle, Washington, 1967, pg. 105. Books available through the author, Mission Hill, SD 57046.

In Hypertension*...When You Need to Conserve K⁺

Every Step of the Way



Each capsule contains 50 mg. of Dyrenium® (brand of triamterene) and 25 mg. of hydrochlorothiazide.

†Step 1 usually consists of an initial phase (a diuretic alone), a titration phase (dosage adjustment and/or addition of a K⁺ supplement or K⁺-sparing agent), and a maintenance phase (a diuretic alone or in combination with a K⁺ supplement or K⁺-sparing agent).

Serum K⁺ and BUN should be checked periodically (see Warnings).

Before prescribing, see complete prescribing information in SK&F Co. literature or PDR. The following is a brief summary.

WARNING

This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K⁺ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K⁺ intake. Associated widened QRS complex or arrhythmia requires prompt additional therapy. Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and tri-

amterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K⁺ frequently; both can cause K⁺ retention and elevated serum K⁺. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia, although uncommon, has been reported. Corrective measures should be instituted

cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function.

Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth, anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis and of impotence have been reported with the use of 'Dyazide', although a causal relationship has not been established.

Supplied: Bottles of 1000 capsules; Single Unit Packages (unit-dose) of 100 (intended for institutional use only); in Patient-Pak™ unit-of-use bottles of 100.

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Motrin[®] vs aspirin w/codeine...

(ibuprofen)



compare the analgesic effect

A Motrin 400 mg dose relieved postsurgical dental pain as effectively as a combination of 650 mg aspirin and 60 mg codeine (two aspirin-with-codeine No. 3 tablets) in a study of 129 patients.

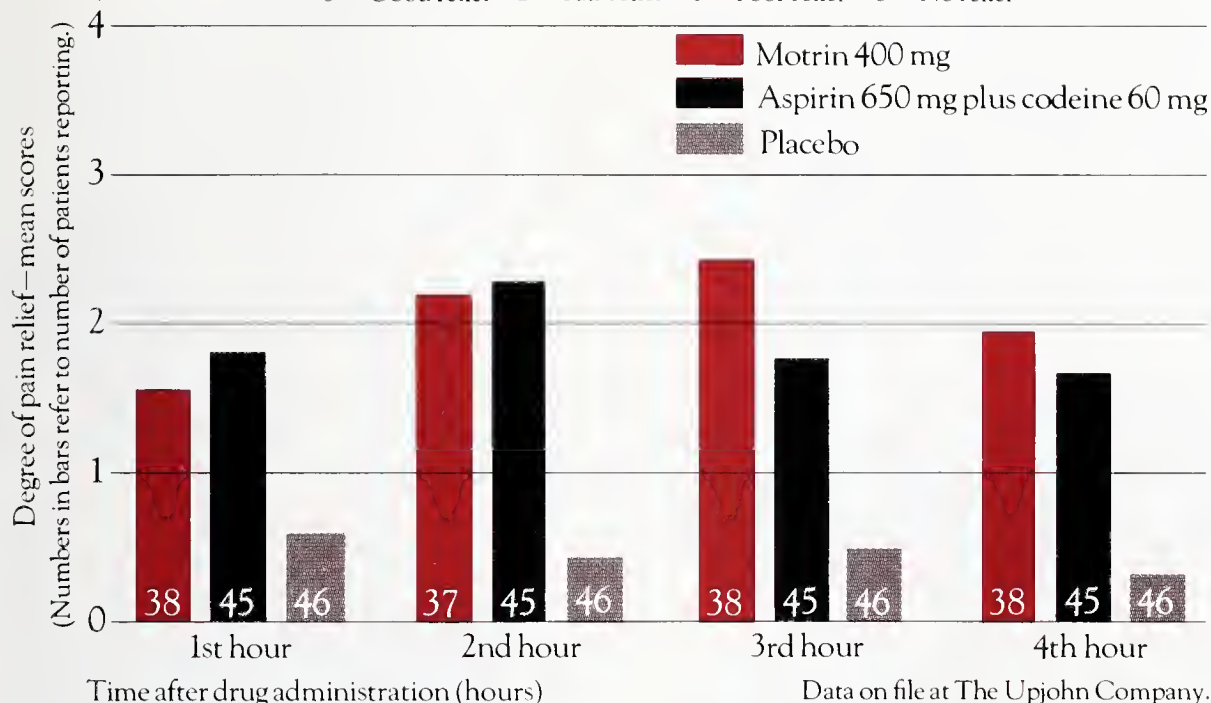
In this double-blind, placebo-controlled, randomized study, no statistically significant difference in relief of pain was noted at 1, 2, and 4 hours between the Motrin and aspirin-with-codeine groups... with Motrin being significantly more effective ($p = 0.03$) at the three-hour interval.

Active treatment was significantly more effective ($p < 0.0001$) than placebo at all time intervals.

Comparison of pain relief

Motrin vs aspirin-codeine combination

4 = Excellent relief 3 = Good relief 2 = Fair relief 1 = Poor relief 0 = No relief



One tablet q4-6h prn

For relief of mild to moderate pain:

Motrin[®] 400mg TABLETS
ibuprofen, Upjohn

- Not a narcotic • Not addictive • Not habit forming • Nonscheduled
- Acts peripherally • Relieves pain rapidly • Relieves inflammation • Indicated in acute and chronic pain • Well tolerated (The most common side effect with Motrin is mild gastrointestinal disturbance.)

Please turn the page for a brief summary of prescribing information.

Upjohn

Motrin[®] (ibuprofen)

now proved an effective analgesic for mild to moderate pain

Motrin[®] Tablets (ibuprofen, Upjohn)

Indications and Usage: Relief of mild to moderate pain.

Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in long-term management. Safety and efficacy have not been established in Functional Class IV rheumatoid arthritis.

Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory agents (see WARNINGS).

Warnings: Anaphylactoid reactions have occurred in patients with aspirin hypersensitivity (see CONTRAINDICATIONS).

Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. Motrin should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If Motrin must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin and the patient should have an ophthalmologic examination, including central visual fields.

Fluid retention and edema have been associated with Motrin; use with caution in patients with a history of cardiac decompensation.

Motrin can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin is added.

Drug interactions. *Aspirin:* Used concomitantly may decrease Motrin blood levels. *Coumarin:* Bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy nor by nursing mothers.

Adverse Reactions

Incidence greater than 1%

Gastrointestinal: The most frequent type of adverse reaction occurring with Motrin is gastrointestinal (4% to 16%). This includes nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of the GI tract (bloating and flatulence). **Central Nervous System:** Dizziness,* headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic:** Decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS).

*Incidence 3% to 9%.

Incidence less than 1 in 100

Gastrointestinal: Upper GI ulcer with bleeding and/or perforation, hemorrhage, melena. **Central Nervous System:** Depression, insomnia. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure. **Special Senses:** Amblyopia (see PRECAUTIONS). **Hematologic:** Leukopenia, decreased hemoglobin and hematocrit.

Causal relationship unknown

Gastrointestinal: Hepatitis, jaundice, abnormal liver function. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities. **Dermatologic:** Alopecia, Stevens-Johnson syndrome. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Hemolytic anemia, thrombocytopenia, granulocytopenia, bleeding episodes. **Allergic:** Fever, serum sickness, lupus erythematosus syndrome. **Endocrine:** Gynecomastia, hypoglycemia. **Cardiovascular:** Arrhythmias. **Renal:** Decreased creatinine clearance, polyuria, azotemia.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

Dosage and Administration: Rheumatoid arthritis and osteoarthritis, including flares of chronic disease: Suggested dosage is 300, 400, or 600 mg t.i.d. or q.i.d. Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for relief of pain.

Do not exceed 2400 mg per day.

Caution: Federal law prohibits dispensing without prescription.

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MED B-4-S

Development Of The South Dakota Psychiatric Association

David W. Bean, M.D.*
William C. Fuller, M.D.**

On May 7, 1978, by action of the Assembly of District Branches of the American Psychiatric Association, the South Dakota Psychiatric Association was officially chartered. The granting of the Charter of the South Dakota Psychiatric Association by its parent association was the culmination of efforts by South Dakota psychiatrists to develop their own district branch of the American Psychiatric Association.

While the South Dakota Psychiatric Society only started in 1978, the history of organized psychiatry which included the state of South Dakota began in May of 1960 when the American Psychiatric Association chartered the development of the North Dakota, South Dakota, and Nebraska Psychiatric Society. That society known as the "Sioux Psychiatric Society" encompassed the very few psychiatrists then practicing in the three bordering states, and continued on in its combined operations until May of 1968. At that time the psychiatrists practicing in the state of North Dakota petitioned for, and received approval to form a district branch which became the North Dakota District Branch of the American Psychiatric Association.

This separation of North Dakota psychiatrists thus reduced the "Sioux Psychiatric Society" membership to those psychiatrists residing in South Dakota and Nebraska who for the most part consisted of Nebraska psychiatrists practicing in the cities of Omaha and Lincoln. The South Dakota psychiatrists, then few in number (7), had little effective

contact with the Nebraska society and virtually no organizational strength.

In October of 1977 at a Psychiatric Department meeting of the USD School of Medicine, inspiration for the development of the South Dakota Psychiatric Association began to spark. At that time it was suggested that practicing psychiatrists in South Dakota should separate from their Nebraska association and form their own separate district branch. There were then the twenty potential members necessary to form a district branch and these persons were contacted to elicit their interest in forming the South Dakota Psychiatric Association. Petitions were developed, a proposed constitution and bylaws were written, and the Nebraska psychiatrists were encouraged to support the development of the South Dakota District Branch. When the petitions, constitution and bylaws were presented to the Assembly of District Branches of the APA, the Nebraska psychiatrists were strong in their support of the South Dakota charter which was granted on May 7, 1978.

The first official act of the District Branch was to elect its officers and did so in a mail ballot in August, 1978. This ballot was officially tallied by the offices of the South Dakota State Medical Association on August 18, 1978.

Table I
Officers as of August 1978

President—David W. Bean, M.D.	Yankton, SD
President-Elect—William C. Fuller, M.D.	Sioux Falls, SD
Representative—David A. Gehlhoff	Sioux Falls, SD
Deputy Representative—Carl N. Rutt, M.D.	Sioux Falls, SD
Secretary-Treasurer—Daniel J. Kennelly, M.D.	Sioux Falls, SD

* Chairman, Department of Psychiatry, USD School of Medicine, Sioux Falls, SD.

**Vice Chairman, Department of Psychiatry, USD School of Medicine, Sioux Falls, SD.

The next official action of the Society was the calling of the first Executive Council meeting on August 30, 1978, held at McKennan Hospital in Sioux Falls. Actions of the Executive Council at that time were to set the first meeting of the Psychiatric Association to be held in Sioux Falls on October 20, 1978; to set the annual dues of \$30.00; to recommend Articles of Incorporation and to develop the agenda for the first meeting of the Society.

The first meeting of the South Dakota Psychiatric Association was called to order at 6:30 p.m., October 20, 1978, at the Howard Johnson Motor Lodge in Sioux Falls, South Dakota. Actions taken at that meeting were the approval of the annual dues of \$30.00 per member, the initiation of ad hoc committees to develop various position statements by the Society, the approval of legal fees for Articles of Incorporation, and the appointment of an Executive Secretary.

Table II
Attendance at First Meeting
October 20, 1978

Charles C. Lord, M.D.	Rapid City, SD
William D. Trumpe, M.D.	Fort Meade, SD
Richard Renka, M.D.	Rapid City, SD
Donald W. Burnap, M.D.	Rapid City, SD
Fred Rosenfeld, M.D.	Hill City, SD
David A. Gehlhoff, M.D.	Sioux Falls, SD
David W. Bean, M.D.	Yankton, SD
William C. Fuller, M.D.	Sioux Falls, SD
Jasper L. Dyer, M.D.	Hot Springs, SD
George A. Richards, M.D.	Sioux Falls, SD
Daryl Stephenson, M.D.	Yankton, SD
Harland T. Hermann, M.D.	Mitchell, SD
Myrick Pullen, M.D.	Yankton, SD

Table III
Twenty Original Members

Leoncio Alonzo, M.D.	Yankton, SD
David W. Bean, M.D.	Yankton, SD
Tajammul H. Bhatti, M.D.	Sioux Falls, SD
Donald W. Burnap, M.D.	Rapid City, SD
Graham H. Chesnut, M.D.	Fort Meade, SD
Jasper L. Dyer, M.D.	Hot Springs, SD
William C. Fuller, M.D.	Sioux Falls, SD
Costas Hercules, M.D.	Rapid City, SD
Harland T. Hermann, M.D.	Mitchell, SD
William D. Trumpe, M.D.	Fort Meade, SD
Robert S. Jones, M.D.	Rapid City, SD
Daniel J. Kennelly, M.D.	Sioux Falls, SD
Richard B. Leander, M.D.	Sioux Falls, SD
Richard Renka, M.D.	Rapid City, SD
George A. Richards, M.D.	Sioux Falls, SD
Fred Rosenfeld, M.D.	Hill City, SD
Carl N. Rutt, M.D.	Sioux Falls, SD
Glen N. Shaurette, M.D.	Aberdeen, SD
Daryl R. Stephenson, M.D.	Yankton, SD
Chung Hao Tuan, M.D.	Yankton, SD

In addition to the routine business, the membership approved the policy that the Psychiatric Society should be an integral part of the profession of medicine and that a strong affiliation between the South Dakota Psychiatric Association and the South Dakota Medical Association should be developed. The attitude of the members then present was, "We are physicians first and then psychiatrists." All members of the Psychiatric Society were encouraged to continue their membership in the South Dakota State Medical Association and to actively participate in the activities of each District and State Medical Society.

Later actions to encourage association with the State Medical Association included the designation of the South Dakota State Medical Association Journal as the official journal of the South Dakota Psychiatric Association, the decision to hold the annual Psychiatric Society meetings in concert with the annual State Medical Association meeting, and to regularly hold a winter meeting of the Psychiatric Society at the same location and in concert with the South Dakota Academy of Family Physicians' annual winter meeting.

The legal mechanisms to develop Articles of Incorporation progressed at a snail's pace, but on June 12, 1980, the South Dakota Psychiatric Association was made "legal" with the receipt and acceptance of the Articles of Incorporation by Alice Kundert, South Dakota Secretary of State.

The purposes of this Psychiatric Society as recorded in Article III of its Articles of Incorporation are:
 "... to advance the medical and collateral sciences and to assist in the acquiring of knowledge of the same; to work for the benefit of community health and welfare; to bring together the physicians who practice psychiatry in this state into one organization and to assist in promoting its aims and objectives; to elevate the standards of medical care in all public programs and to advise persons or agencies in the administration of such programs;"

The Society has continued to grow with 27 members as of February 20, 1981, and successful efforts to improve the over-all mental health care of the citizens of the state of South Dakota have been mounted. Some significant accomplishments are: 1) the recruitment of six fully trained and licensed psychiatrists to the South Dakota Human Services Center, 2) the subsequent upgrading of several Human Services Center treatment programs; 3) the recruiting of two psychiatrists based primarily in Community Mental Health Centers outside of the urban areas; 4) the recruiting of four child psychiatrists to the state of South Dakota; and 5) the establishment of an inpatient psychiatric unit at the Rapid City

Regional Hospital.

Position papers developed by the Psychiatric Association include the following: 1) the position that citizens of South Dakota should receive health insurance with equal coverage of psychiatric illness as other medical illness; 2) the position that electroconvulsive therapy is a safe and essential mode of psychiatric treatment for certain emotional illnesses; 3) the position that all mental health professionals should be licensed according to appropriate licensing body standards; 4) that all public mental health facilities should meet minimum national standards of professionalism and fire safety, and 5) the position that there are certain "patient rights" for individuals receiving mental health services in the state of South Dakota.

Current efforts are focusing on adequate reimbursement for mental health services, public relations' efforts for psychiatry in South Dakota, and recruiting efforts. Psychiatry, as a medical specialty, has been and continues to be, furthered by the development of the South Dakota Psychiatric Association and the pioneering efforts of its early leadership.

The South Dakota State Psychiatric Association, while one of the youngest members of the medical specialty societies affiliated with the South Dakota State Medical Association, pledges its continued fellowship with other physicians and its continued activities to support improved mental health services to the citizens of South Dakota and the United States.



William R. Taylor, M.D., SDSMA Delegate to the AMA from 1976-1980 being interviewed at an AMA meeting.



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The History Of Radiology In South Dakota

Donald H. Breit, M.D.*

James F. Wunder, M.D.**

Radiology was introduced into South Dakota in the gas tube era by Nelius J. Nessa, M.D.

Dr. Nessa was graduated from the University of Minnesota School of Medicine in 1904. After internship and brief general practice in Brewster, Minnesota, he moved to Sioux Falls and joined Dr. R. G. Stevens, a general surgeon in operating Good Samaritan Hospital. He became interested in radiology and took a course from Dr. James Case of Chicago in 1915. He returned to Sioux Falls and in association with Drs. E. E. Gage and Guy Van-Demark, continued his general practice but devoted as much time as possible to x-ray work. He used a gas tube in a lead glass bowl energized by an induction coil. In 1918 he enlisted in the U. S. Army and had further training in radiology at Fort Oglethorpe in Georgia and at Great Lakes Training Station, Chicago.

After his discharge in 1920 he was a member of a group that organized the Sioux Falls Clinic. He limited his practice to diagnostic and therapeutic radiology and became the first specialist in radiology in South Dakota. He was joined in July of that year by W. S. Beckstrom, a well trained technician who had studied at the army medical school in Johns Hopkins Hospital.

The X-ray Department in the Sioux Falls Clinic was quite well furnished with the most modern and up-to-date equipment of that time. Two complete Schnook units were installed, a 180-KVP for intermediate and deep therapy and a 135-KVP for diagnostic procedures and superficial therapy. Coolidge tubes in open lead bowls were used overhead for

diagnosis and therapy, and a tube beneath the table served for fluoroscopy. An upright fluoroscope was installed in a curved table with a curved Bucky for radiographic work completed the installation.

Dr. Nessa was radiologist for both Sioux Valley and McKennan Hospitals. Films were taken at the hospitals and in homes by Mr. Beckstrom. He used a Scheidel-Western gas tube unit, suitcase type with a portable tube stand. Later a Coolidge model F portable unit was used. The films were developed at the Sioux Falls Clinic and interpreted by Dr. Nessa.

In 1928 St. Luke's Hospital in Aberdeen was opened with Dr. Paul V. McCarthy as the radiologist. He was the second physician in South Dakota doing radiological work as a specialty. Dr. McCarthy graduated from St. Louis University Medical School and became interested in radiology as an intern in St. Louis City Hospital by Dr. L. R. Sante. Dr. Sante had established himself in St. Louis as a radiologist after World War I. Dr. McCarthy returned to St. Louis before 1928 and spent some time working with Dr. Sante in St. Louis City Hospital. During the early 30's, Dr. McCarthy found it necessary to augment his radiological practice with general practice. During that era, the practice of non-radiologists doing x-ray work to some extent was established and has continued to the present time. Dr. McCarthy used similar equipment as Dr. Nessa. In 1939, shock-proof equipment was installed in the hospitals in Sioux Falls and Aberdeen. By that time, radiology was well established as a specialty in eastern South Dakota under the leadership of Drs. Nessa and McCarthy. Dr. McCarthy died in the late 1970's.

The western part of the state became the next focus of radiology development with Dr. J. Richard Fuchlow who became the first radiologist to serve the Black Hills area as a specialist. He established his practice in Rapid City in 1941. He had been an x-ray

* Radiologist at Sioux Valley and McKennan Hospitals and consultant at the V. A. Hospital, Sioux Falls, SD.

**Radiologist at Mobridge Community Hospital, Holy Infant Hospital, Hoven, Bowdle Hospital and Faulk County Hospital.

technician who studied medicine, received his medical degree and specialized in radiology. He retired in 1944 and died of a coronary occlusion.

Dr. Ernest H. Brock, a Texan, replaced Dr. Fuchlow in 1944, opening an office in the old Black Hills General Hospital. In 1947 he opened a private office but continued to serve both Rapid City Hospitals as well as the Veterans Hospital at Fort Meade and St. Joseph's Hospital in Deadwood. In the latter part of 1950, he suffered a coronary occlusion and retired in Texas. Drs. Alton J. Saxton and George F. Wood, Jr. took over his practice. Dr. Saxton was from Lincoln, Nebraska and Dr. Wood from St. Paul, Minnesota. Both were certified radiologists and continued Dr. Brock's practice serving the same hospitals. In addition, they extended radiological services to Homestake Hospital in Lead, the Veterans Hospital in Hot Springs and the community hospitals of Spearfish and Belle Fourche. They were joined in 1957 by Dr. John Hewitt.

Dr. Hans Jacobi was another pioneer radiologist in eastern South Dakota locating in Huron. He received his medical degree in Germany and was trained in radiology in Breslau. He started in Huron in May, 1943 and was the first specialist in radiology in that community. He was a member of the staff of the Huron Clinic and Sprague Hospital, and at St. John's Hospital when it opened in 1947. He died in 1958 at the age of 55. Huron was without radiological services until February, 1960 when Dr. G. M. Huet established practice in Huron. During the latter part of World War II Dr. Marianne Wallace, a fine foreign trained radiologist, assisted Dr. Nessa. In 1946 Dr. Donald H. Breit of St. Joseph, Missouri joined Dr. Nessa in Sioux Falls and became the sixth radiologist to engage in specialty practice in South Dakota. He received his medical degree from Baylor University, was trained in surgery, and was in surgical practice before entering the field of radiology. He completed his three year residency program in radiology at the University of Nebraska Hospital under the direction of Dr. Howard B. Hunt.

In July, 1948, Dr. C. Stanley Larson, a native of Sioux Falls joined Dr. Nessa and Breit in partnership practice of radiology. Dr. Larson trained at the Lahey Clinic but came to Sioux Falls from a practice in Corpus Christi, Texas. Dr. Nessa died in 1948 and the practice continued by Drs. Breit and Larson. Dr. Donald J. Peik of Minnesota joined the partnership in July, 1951, after completing his residency in Milwaukee. Dr. Bryson R. McHardy, also from Minnesota, joined the partnership January 1, 1959, after completing his residency in Chicago.

Yankton, South Dakota was introduced to modern radiology in July, 1949 by Dr. James P. Steele. Dr. Steele received his radiological training at

Louisville General Hospital, Mallinckrodt Institute of Radiology, St. Louis and St. Joseph's Mercy Hospital, Ann Arbor, Michigan. In March, 1950, Dr. Saul Friefeld settled in the Brookings area and provided the first regular radiological service to Brookings, Madison, Flandreau, Lake Preston, Volga, and Estelline. The capitol city of South Dakota, Pierre, was one of the last major cities in the state to be served by a specialist in radiology. Dr. Hubert E. Werthmann located there in 1959. He was a native of Germany, receiving his medical education there but had his training in radiology under Drs. Leo Rigler and H. O. Peterson at the University of Minnesota Hospital as a Mayo fellow. Dr. Warren S. Peiper began radiology practice in Mitchell, one of the larger cities in Eastern South Dakota, in November, 1953. He soon extended his services to Parkston and Chamberlain. Watertown was the last of the larger communities in South Dakota to have the services of a radiologist as a specialist. Dr. Charles F. Ryan established practice in August 1, 1957. He had received his training at Albany and came to the state immediately following his residency. Dr. Ryan died in 1979.

Radiology has progressed steadily in South Dakota since 1964. The introduction of image amplifiers in the 1960's has tremendously upgraded the image quality of fluoroscopy and has seriously refined diagnostic efforts. During the late 1960's, angiography was introduced into the major medical centers in the larger cities and has continued to grow as a needed diagnostic tool for specialty groups. Nuclear medicine came into its own recognition in the early 60's for diagnostic and therapeutic utilization, and today is represented in almost all areas served by a radiologist. This modality extended the scope of diagnosis and therapy available to the medical community. Radiation therapy has progressed from the early efforts of x-ray therapy utilizing the early 180-KVP units of Dr. Nessa and later units of the 250-KVP x-ray therapy to the modern use of cobalt and linear accelerators for better and more efficient deep therapy of tumors. Ultrasound is a relative newcomer to the imaging modality of radiology that has gained wide acceptance—causing no ionizing radiation damage to living tissue and continues to grow and become more refined. B mode ultrasound with Grey Scale imaging has been improved with the advent of Real Time Ultrasound and sector scanning which have been coupled with computers to give excellent diagnostic imaging to radiologists and physicians across the state.

A significant change in radiology practice occurred in the late 1960's with the introduction of high dose intravenous contrast examinations which tremendously improved IVP's, venograms and ar-

tertiograms. Coupled with newer and less toxic contrast agents, image quality was improved with less patient morbidity. The most significant addition to the imaging profession came with the introduction of computerized axial tomography (CAT) scanning in the early to mid 70's which has brought radiology into the forefront of medical specialties. As with all new procedures there has been considerable controversy with the adaptation of x-ray use for diagnosis, but mostly caused by high initial cost and government interference through the certificate of need restriction limiting their acquisition and use. Sioux Falls installed the first unit in the state using a Delta 50 unit which was upgraded two years later to an Ohio Nuclear 2000 unit, giving a scan time of approximately two seconds per image.

In recent years, several radiologists have come to South Dakota. The trend has been to practice in groups whenever possible rather than individual radiologists as was the practice in the past. The largest group in the state is in Sioux Falls with twelve mem-

bers in the group and the next largest in Rapid City with eight members. Whenever possible, two or more radiologists will be practicing together except in the smaller communities when this is not possible. Most of the communities in the state have radiological coverage of some type. As of 1981, there are 36 practicing radiologists in the state, 33 of which are certified by the American Board of Radiology.

Several teaching programs have been established for training technologists, however, more students apply than can be accommodated at the present time.

The progress of radiology in South Dakota since its rather recent beginning in 1915 has been quite substantial. The past twenty years has shown the growth of radiology, in a primarily rural state, has developed into a modern efficient specialty of which all radiologists and the medical community can look upon with pride and satisfaction.



The Grievance Committee of the SDSMA met in 1958 during the annual meeting held in Huron, and included from left to right: Alonzo Peeke, M.D., Chairman Louis Pankow, M.D., David Gregory, M.D. and Arthur Spiry, M.D.

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Use in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

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Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported, should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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Pediatrics In South Dakota

W. F. Stanage, M.D.*

The first recorded mention of an ill child preceding white settlement in the land of Dakota was in 1680. Father Louis Hennepin, while exploring the area of the Upper Mississippi River was taken to an Indian cabin where he found a sick child. The child was baptized Antoinette, but died soon after.

With white settlement in the late 1850's and early 1860's, several doctors came to the territory. Dr. Justus Townsend came to Yankton and soon found himself "not entirely busy" with his medical practice. He was Secretary of the first Congress, Auditor, and County Commissioner. Dr. Phillips came to Sioux Falls with one of the land companies and did town-site promotion, in addition to practicing medicine.

The practice of medicine was rudimentary at its best. Illnesses relating to children were so often fatal that there were few cures and even less prevention. The survival rate of children had not improved much from colonial times when one-third of the children born did not live to be ten years old.

Newspaper accounts in the early days were a typical forecast of pediatric disasters. The weekly **Dakotian** of Yankton on June 17, 1862, reported that "the little community of Brule Creek settlement was shocked by the sudden death of Susan Oleson, a Norwegian girl of fourteen years of age. Dr. Phillips was hastily called, and every appliance possible was used to restore life, but in vain." On July 5, 1862, the paper reported that "the little daughter of Honorable H. D. Betts died of diphtheria a few days ago." Death continued from relapsing fever, diphtheria, bilious colics, smallpox, scarlet fever, internal fever, whooping cough, typhoid fever, infantile cholera, and consumption.

Pediatrics as a specialty was first introduced in

America by Abraham Jacobi who taught pediatrics in New York City from 1857 to 1899. He was followed by Job Smith, L. Emmett Holt, and Thomas Rotch. Even with an increasing knowledge and a growing interest in preventive medicine, it was not until the beginning of the Twentieth Century that the emerging specialty of pediatrics began to have an impact on the health and well-being of children.

The first pediatrician in South Dakota and a Charter member of the American Academy of Pediatrics was Goldie Eleonora Zimmerman. Dr. Zimmerman was born in St. Charles, Minnesota in 1887. She had attended high school in Aberdeen and was a graduate of the University of Illinois on June 6, 1911. She was licensed to practice medicine in South Dakota on July 13, 1911. She then came to Sioux Falls in the practice of pediatrics in 1915. She practiced in Sioux Falls until 1943.

The next pediatrician to practice in South Dakota was Dr. William Emmett Donahoe. Dr. Will, as he was affectionately called, was born May 18, 1886, in Sioux Falls, Dakota Territory. He attended school in Sioux Falls and The College of St. Thomas in St. Paul. In 1912 he graduated from Medical School of the University of Illinois in Chicago. Following internships in Chicago and Minneapolis, he returned to Sioux Falls and practiced general medicine from 1914 to 1919. Dr. Donahoe then studied pediatrics at the University of Iowa and in Chicago and Minneapolis. He returned to Sioux Falls in 1920 and limited his practice to pediatrics.

Dr. Donahoe was the first in the state to introduce immunizations for diphtheria, tetanus, and pertussis. He encouraged many aspects of preventive medicine, including diet, vitamins, and smallpox immunization. He was active in establishing clinics for sick and well infants. Many of these were held at state and county fairs where he converted "cutest

*Pediatrician, Yankton Clinic, Yankton, SD.

baby" shows into Infant and Child Clinics, stressing examinations and immunizations.

At a meeting in Sioux Falls, chaired by Dr. H. W. Farrell, on June 2, 1964, the formation of a Pediatric Society was discussed, and initial steps for its founding were started. Dr. Farrell was elected temporary president, and Dr. E. H. Heinrichs, secretary. Present at the meeting were Drs. H. W. Farrell, Warren Anderson, N. J. deDianous, Will Donahoe, W. F. Stanage, G. E. Tracy, and E. H. Heinrichs. Dr. W. F. Stanage was elected chairman of the American Academy of Pediatrics and the newly formed Pediatric Society in 1967. A constitution and by-laws of the South Dakota Chapter of the American Academy of Pediatrics and the South Dakota Pediatric Society were presented to the pediatric group at the

May 1968 State Medical Society meeting. Due to the absence of many potential members, action was delayed. The constitution and by-laws were adopted and accepted by mail vote in 1969. The roster of the members of the Society in 1969 included twenty physicians; ten were members of the Academy of Pediatrics. Dr. Nat Whitney was elected chairman of the State Chapter of the Academy and the Pediatric Society in 1973, and Dr. E. H. Heinrichs in 1979.

The South Dakota Chapter of the American Academy of Pediatrics and the South Dakota Pediatric Society continues to tend to the health of children, following the example of Dr. Will Donahoe. There are at present fifty members in the organization, and thirty are Fellows of the American Academy of Pediatrics.



Survivors of the massacre on Wounded Knee Creek in 1890 were treated in this makeshift hospital at Pine Ridge Indian Agency, South Dakota. One physician who didn't fare well in the history was Dr. Daniel F. Royer, the Indian agent at the time of the bloody carnage. Woefully inexperienced, he was nicknamed by the Sioux Young-Man-Afraid-of-Indians. He lost control on the reservation, and his political plum became a bitter lemon.

Reprinted from *Doctors of the Old West* by Robert Karolevitz, Superior Publishing Co., Seattle, Washington, 1967, pg. 43. Books available through the author, Mission Hill, SD 57046.

History Of South Dakota Society Of Internal Medicine

The formation of the South Dakota Society of Internal Medicine was indeed a "grassroots" development. It was, and remains, based on the concerned interest of physicians in an organization for the furtherance of the practices of Internal Medicine; to study the scientific, economic and social aspects of medicine in order to secure and maintain the highest standards of practice and the best possible patient care; and to share scientific knowledge in order to secure and promote adequate community health and welfare.

The initial organizational meeting was held at the Huron Clinic (hosted by C. F. Gryte, M.D., and B. T. Lenz, M.D.), on September 15, 1951, with twenty interested South Dakota physicians participating (including representatives in pathology, pediatrics and dermatology). The officers elected were President J. L. Calene, M.D., of Aberdeen, Vice-President, C. F. Morsman, M.D., of Hot Springs; and Secretary-Treasurer, T. H. Sattler, M.D. of Yankton. The scientific program was presented by D. S. Berkman, M.D., on the etiology of arthritis; I. L. Schuchardt, M.D., on the differential diagnosis of coronary occlusion and hiatus hernia; and T. H. Sattler, M.D., on delayed EKG changes in myocardial infarction and a report on familial infectious hepatitis.

Spurred on by the success of the first meeting, subsequent successful yearly meetings were held with active participation of the members of the Society in the scientific part of the program. These scientific programs were stimulated and encouraged by the American College of Physicians (founded in 1915). Representing the College in those early formative years as Governor were John L. Calene, M.D., of Aberdeen, followed by C. F. Morsman,

M.D., of Hot Springs, and D. L. Kegaries, M.D., of Rapid City. In the early years, the associated business meetings dealt with such matters as (1) eligibility criteria for Society members, (2) drawing up a Constitution and By-laws, (3) working out Blue Shield problems, and (4) encouraging liaison with other specialty groups in South Dakota.

In 1957, the South Dakota Society affiliated with the American Society of Internal Medicine, a national organization, which had been formed in 1957 to upgrade the status of the Internist and encourage him to become more knowledgeable and efficient in medico-economic matters. In 1958, the Constitution and By-laws of the South Dakota Society of Internal Medicine were changed to correspond with those of ASIM.

On September 20, 1960, at the University of South Dakota Medical School in Vermillion, South Dakota, the first **combined meeting** of the South Dakota membership of the ACP and ASIM was held. Since that time, one and one-half day meetings have been held annually in September, with the meetings alternating yearly among Sioux Falls, Rapid City, and Vermillion (or Yankton).

On November 22, 1960, the South Dakota Society of Internal Medicine was incorporated as a non-profit organization. Trustees designated in the Articles of Incorporation were R. F. Thompson, M.D.; D. R. Driver, M.D.; G. S. Paulson, M.D.; T. H. Sattler, M.D.; J. L. Calene, M.D.; and D. L. Kegaries, M.D.

New arrivals in the state have been encouraged to participate in our annual meetings and to become active members of both ACP and ASIM. Representatives of the parent organizations are present at each annual meeting. From the initial nucleus of

twenty members, the organization has grown to a potential of almost one hundred members. Though small in number, the SDSIM has prided itself on the number of its members who have made generous contributions in time and talent to the State Medical Association and to a variety of other medically related organizations, such as PSRO, Blue Shield Board, South Dakota School of Medicine, Regional Medical Programs, Health System Agencies, etc. In addition, many have been involved in numerous community organizations and activities.

Even though the character of the Society has changed somewhat, in that most members in 1951 were general Internists and now many are in the

sub-specialty fields or are exclusively in academic medicine, the goal of the organization remains the same. That goal is to maintain the highest possible standards of ethical and scientific achievement possible, always keeping in mind that our ultimate purpose is to provide the best possible patient care, within practical reason.

In 1979, the Society awarded its Distinguished Internist Award to D. L. Kegaries, M.D., of Scottsdale, Arizona; in 1980, a similar award was presented to T. H. Sattler, M.D., of Yankton, South Dakota. A listing of Past-Presidents of the Society and the Governors of South Dakota for the American College of Physicians follows:

Table 1
Presidents Of The South Dakota Society Of Internal Medicine

Name	Location	Year
*John L. Calene, M.D.	Aberdeen	1951-1953
*Charles F. Morsman, M.D.	Hot Springs	1953-1954
Theodore H. Sattler, M.D.	Yankton	1954-1955
John W. Donahoe, M.D.	Sioux Falls	1955-1956
Don L. Kegaries, M.D.	Rapid City	1956-1957
Warren L. Jones, M.D.	Sioux Falls	1957-1958
Mary E. Sanders, M.D.	Redfield	1958-1959
Robert F. Thompson, M.D.	Yankton	1959-1960
Donn R. Driver, M.D.	Sioux Falls	1960-1961
Gordon S. Paulson, M.D.	Rapid City	1961-1962
William R. Taylor, M.D.	Aberdeen	1962-1963
Everett W. Sanderson, M.D.	Sioux Falls	1963-1964
Carroll J. Clark, M.D.	Watertown	1964-1965
John W. Argabrite, M.D.	Watertown	1965-1966
Reuben J. Bareis, M.D.	Rapid City	1966-1967
*Clark F. Johnson, M.D.	Yankton	1967-1968
Robert J. Ogborn, M.D.	Sioux Falls	1968-1969
H. Streeter Shining, M.D.	Rapid City	1969-1971
William O. Rossing, M.D.	Sioux Falls	1971-1973
Mary E. Sanders, M.D.	Aberdeen	1973-1975
Robert K. Johnson, M.D.	Rapid City	1975-1977
Lynn I. DeMarco, M.D.	Sioux Falls	1977-1978 (Jan.)
William W. Quick, M.D.	Yankton	(Jan.) 1978-1979
O. Myron Jerde, M.D.	Rapid City	1979-Present

*Deceased

Table II
ACP Governors For South Dakota

Name	Location	Year
*John L. Calene, M.D.	Aberdeen	1937-1949
*Charles F. Morsman, M.D.	Hot Springs	1949-1958
Don L. Kegaries, M.D.	Rapid City	1958-1964
Theodore H. Sattler, M.D.	Yankton	1964-1970
Gordon S. Paulson, M.D.	Rapid City	1970-1971
Robert F. Thompson, M.D.	Yankton	1971-1976
Everett W. Sanderson, M.D.	Sioux Falls	1976-1980
Reuben J. Bareis, M.D.	Rapid City	1980-Present

*Deceased

A Brief History Of Pathology In South Dakota

Peter Norbeck Wegner*

Karl H. Wegner**

Ben E. Diamond***

Joseph C. Ohlmacher, M.D.
 "The Father of SD Pathology"
 Professor of Pathology & Bacteriology 1918-1933
 Dean, School of Medicine 1933-1946
 The University of South Dakota
 First Director-State Health Laboratory
 President, South Dakota State Medical Association 1944



A history of pathology in South Dakota is not an easy task to approach: already the passage of time has partially obscured its origins and the authors are too close to its present development to offer the objectivity that only time and detachment can provide. While it is impossible, therefore, to clearly define the beginning of pathology in South Dakota, its evolution closely parallels the development of medical education and private practice within the state.

Dr. Joseph Christian Ohlmacher is generally acknowledged as "The Father of Pathology in South Dakota". A graduate of Rush Medical College, he joined the youthful medical school in 1918, eleven years after classes commenced. Ohlmacher served for many years as Professor of Pathology and Bacteriology and as the first director of the State Health Laboratory. His professional activities comprised primarily autopsy and surgical pathology: duties which he performed for a substantial part of the

state, including the Sioux Falls and Yankton hospitals. Serving as dean of the medical school, Ohlmacher contributed his expertise to the old basic sciences school from 1933 to 1946. In 1944, the South Dakota State Medical Association elected him president, where he further demonstrated his administrative ability. Ohlmacher practiced for several more years as the first pathologist at St. Luke's Hospital in Aberdeen.

Ohlmacher's position of Pathology Department Chairman was subsequently filled by several capable educators. Among his successors was Dr. Amos E. Michael, remembered with respect and affection by the medical students as "Black Mike". Michael chaired the Pathology Department from 1951 to 1960. Dr. George W. Knabe, Jr., served as chairman from 1961 to 1967, and as dean from 1968 to 1972. Dr. Karl H. Wegner followed Knabe as chairman from 1968 to 1972 and as dean from 1972 to 1979. Wegner, serving concurrently as the University's first vice president for health affairs, helped to guide the medical school from its traditional two-year basic science status into a full four-year degree-granting program. After witnessing the graduation of the first three classes of M.D.'s, Wegner returned to private practice.

* Senior, Lincoln High School, Sioux Falls, SD.

** Professor of Laboratory Medicine, University of South Dakota; Medical Director, Laboratory of Clinical Medicine (LCM), Sioux Falls, SD.

***Adjunct Professor, Gardner-Webb College, Boiling Springs, NC; Director Emeritus, SD State Health Laboratory.

The roots of fulltime private practice are divided between two fine pathologists who came to the state in 1947: Dr. John T. Tidd, who practices in Yankton, and Dr. Wayne A. Geib, who practiced in Rapid City. In 1955, Dr. John Elston joined Dr. Geib in his practice and served with distinction as president of the SDSMA in 1969. Dr. Durward Lang of Sioux Falls, the current vice president, would become only the third pathologist to assume the presidency.

The pathologists have long played a leading role in the only continuing professional medical publication in the state, the **South Dakota Journal of Medicine**. Dr. Albert M. Harris, an early pathologist at Sioux Valley Hospital, reported his research on a new cancer detection test in the first issue in January, 1948. Although long since invalidated, the test created some initial nationwide publicity after it showed a positive result on Harris himself, who died of a glioma a few months later (**TIME** magazine, February 9, 1948). Dr. John F. Barlow has given enormous and continuous professional credence to the Journal with his numerous contributions. Pathologist at the Laboratory of Clinical Medicine and Sioux Valley Hospital, and professor of laboratory medicine at the medical school, Barlow's near-monthly reports of "The Clinicopathological Conferences of Sioux Valley Hospital" have been an integral part of the Journal since 1966.

The educational aspect of the development of pathology in South Dakota took another form with the South Dakota Society of Pathologists. Formed shortly after World War II, the society began with informal and infrequent meetings including Dr. Geib and other pathologists. Dr. Arnold K. Myrabo, a widely-respected professional and Chief of Pathology at McKennan Hospital, held the first organizational meeting at his home in the spring of 1952. Dr. Charles B. Mitchell of Sioux Falls and Drs. Tidd and Michael also attended. Since 1952, the Society has held annual or bi-annual meetings to discuss both professional and socioeconomic aspects of practice. In more recent years, meetings have been held at the time of the State Medical Association convention. Dr. Richard D. Schultz of Sioux Falls is the current president.

Pathologists in South Dakota have also taken active roles in more specifically student-oriented educational programs. From undergraduate college education to undergraduate medical education, through graduate and post-graduate education, pathologists in South Dakota have provided invaluable leadership and assistance.

Several hospitals in the state have sponsored, through the aegis of their pathologists, approved

schools of medical technology. Through these programs, fourth-year college students have the opportunity to take a hospital-based internship in medical technology.

For more than half a century, pathologists have been involved with formal undergraduate medical education with the USD medical school. In more recent years, pathologists throughout the state have continued and reinforced this precedent by teaching in the medical school curriculum. Dr. Richard A. Jaqua of Sioux Falls has encouraged and supported this participation in his capacity as Chairman of Laboratory Medicine, developing a statewide base for his department.

Pathologists, in addition, have long been associated with programs of graduate medical education in other specialties in South Dakota. Formal graduate medical education in pathology alone, however, began more recently with a four year residency program initiated by Dr. Mitchell in about 1960. Dr. Wegner resumed this Sioux Valley Hospital-based program in 1964, where it has continued without interruption. For the past decade, it has been under the leadership of Dr. Barlow and is an actively affiliated program of the medical school.

The LCM Foundation has recently instituted a unique program in post-graduate pathology education in the form of a post-doctoral fellowship. The first recipient, Dr. Theodore M. Bailey, Jr., was a summa cum laude graduate of the University of California. Bailey spent the year immersed in a program of post-doctoral education, punctuated with periodic visits to the laboratories of the Public Health Service Hospitals on the state's Indian reservations.

While the education aspect of pathology in South Dakota is undoubtedly important, an equally interesting aspect of its growth and development is the private practice of so many competent and dedicated pathologists across the state.

During a period when the practice of pathology nationwide has often narrowed to a more restricted basic science or research approach, the practice of pathology in South Dakota has been enriched by an ever-broadening clinical base. Several of the state's pathologists have separate certification by the American Board of Nuclear Medicine, devoting much of their time to practice in nuclear medicine and nuclear cardiology. Others spend much time in hematology, ultrasonography, virology, or electron microscopy at a time when such professional activities elsewhere are often performed by other specialists.

History Of The South Dakota State Department Of Health (1891-1950)

The South Dakota State Board of Health was created by legislative act in 1891 and consisted of three physicians appointed by the Governor. The original members of the Board were Doctors C. B. Alford, W. C. Fowler, and D. W. Robinson. Dr. Alford was designated by the Governor as the first Superintendent of Health and he was also elected by the Board as its first President. The position of Superintendent of Health and President of the Board was an honor extended successively to each of the three original members. The sum of \$1,000 annually was appropriated by the Legislature for maintenance of the Board; this was decreased two years later to \$500.

The first meeting of the State Board of Health was held in Aberdeen on July 9, 1891, and at this time county boards of health were organized, each board consisting of two physicians, one of whom was designated as Superintendent, and the State Attorney who was ex-officio president of the board.

In 1893, the State Board of Health was designated as a board of examiners for the purpose of licensing physicians desiring to practice medicine and surgery in the state. Most of the Board's time in the earlier years of its existence apparently was given to this activity. There were many instances of investigation of practitioners who were practicing without license or who had received a license on the basis of fraudulent credentials. Written examinations for licensure were not required and a license was issued upon presentation of a diploma from a medical college indicating that four courses of study of not less than six months each had been successfully completed. There being no provision made for the accrediting of medical colleges, it can readily be seen why "diploma mills" did such a flourishing business.

The first public health action to be taken by the State Board of Health was in connection with a flock of sheep in Clark County which was reported to be infected with "temta fimbriata". Quarantine of the flock was ordered pending action at the next meeting. At that time an order was issued that the sheep be destroyed. The first regulations adopted by the Board were in the field of medical licensure and defined what was considered to be unprofessional conduct which would warrant revocation of license. The activities enumerated are practically the same as those still specified in current medical practice acts.

The first regulations relating to communicable disease control were adopted in 1892. These dealt largely with the control of scarlet fever, diphtheria, and smallpox and in many respects sound principles of control were set forth which are still recognized today in the prevention of the spread of these diseases. The importance of vaccination against smallpox was recognized, as well as proper isolation of the patient, concurrent disinfection, and terminal disinfection. Not in keeping with present day knowledge were provision for fumigation of sick rooms by the fumes of burning sulphur and the warning to avoid by all means the inhaling of the desquamated scales from the skin of a scarlet fever convalescent.

The primary health problem in South Dakota at the turn of the century was the prevalence of smallpox. In spite of resolutions by the Board of Health urging universal vaccination, cases occurred by the hundreds and often by the thousands annually and no section of the state was spared. The acuteness of the problem is indicated by a report of an epidemic of smallpox which occurred in Sisseton and the surrounding area in Roberts County in 1901. An investigation by the Board revealed the presence of 175

cases of smallpox. Nineteen townships were immediately placed under quarantine and all public gatherings were prohibited. Vaccination was ordered for all in the area, with isolation the penalty for all of those not complying. No mail, express, or freight was permitted to be taken from the quarantined area. That this rather heroic action was successful in achieving its purpose is indicated by the fact that twenty-one days later, when quarantine was lifted, there were two thousand persons in the area who had been vaccinated and no further cases occurred.

In 1903 legislation was passed clarifying and extending the powers and duties of the State Board of Health. Provision was made for a five member board. In 1904, a need was expressed for the collection and recording of vital statistics and in 1905 a state agency for this purpose was set up in the office of the State Historian. In 1920, the Bureau of Vital Statistics was transferred to the State Board of Health where it has remained since.

Interest in laboratory work as an adjunct to communicable disease control was first noted in 1909. At this time, provision was made at the State University Bacteriological Laboratory for the free examination of specimens for the diagnosis of diphtheria, tuberculosis, typhoid and anthrax. The examination of brains of suspected rabid animals and specimens of water from public water supplies were also provided at no charge. The records show that the number of laboratory examinations previous to 1916 averaged less than 1000 per year. This is in contrast to 160,000 such examinations now being made in the state annually.

In 1913 provision was made for the office of a full time Superintendent of the State Board of Health. P. B. Jenkins, M.D., was appointed to this office, a position which he held almost continuously for a period of twenty-five years until December, 1938. Succeeding Dr. Jenkins as full time Superintendents have been the following:

- J. F. D. Cook, M.D., December, 1938 to February, 1943
- Gilbert Cottam, M.D., May, 1943 to July, 1948
- G. J. Van Heuvelen, M.D., July 1948 to present

Dr. Jenkins established the office of the Board in Waubay and it remained there until 1933 when it was moved to the Capital building in Pierre, its present location.

The original State Board of Health had two functions, medical licensure and communicable disease control. Other activities and new programs have been added from time to time as follows:

Records and Accounts	1913
Vital Statistics	1920
Public Health Nursing	1920
Sanitary Engineering	1921

Public Health Education	1921
Maternal and Child Health	1922
Epidemiology	1926
Crippled Children's Services	1936
Venereal Disease Control	1938
Division of Laboratories	1939
Dental Health	1946
Tuberculosis Control	1945
(Federally sponsored)	
Cancer	1947
(Federally sponsored)	
Mental Health	1947
(Federally sponsored)	
Heart Disease	1947
(Federally sponsored)	
Hospital Facilities	1947
(Federally sponsored)	

In 1947 recognition was given to the fact that the old Board of Health organization was no longer in keeping with the actual administration of public health programs in the state. By legislative act, a State Department of Health was created. Replacing the old Board of five physicians is a Public Health Advisory Council of nine members representing not only the medical field but the other professions interested in health. Provision is made for the establishment of Divisions within the Department under the direction of qualified technical personnel. It is required that the State Health Officer shall have had special training and experience in the field of public health.

It is interesting to note that in 1900 the official state health agency consisted of three physicians who met occasionally as conditions seemed to indicate. The property owned by the Board consisted of a letter press, a seal, and a small assortment of record books and blanks. The annual budget was \$750. This is in contrast with the State Department of Health in 1950 which employs 109 full time personnel, 66 in professional and 43 in clerical positions, owns equipment valued at approximately \$81,000, and operates on an annual budget of \$657,729, of which \$131,418 is appropriated by the State and \$526,311 represents grants from Federal health agencies and funds made available by counties for local health services.

A History Of South Dakota Blue Shield

South Dakota Blue Shield had its beginning on June 1, 1956. On that date, Articles of Incorporation were filed in Sioux Falls.

Charter members of the Corporation were C. J. McDonald, M.D.; A. A. Lampert, M.D.; Robert Monk, M.D.; P. H. Hohm, M.D.; E. A. Johnson, M.D.; and D. H. Breit, M.D.

The first annual corporate meeting also was held on June 2, 1956, with the following individuals elected to serve on the Board of Directors:

C. J. McDonald, M.D., Sioux Falls
Robert Monk, M.D., Yankton
D. H. Breit, M.D., Sioux Falls
E. A. Johnson, M.D., Milbank
P. H. Hohm, M.D., Huron
A. A. Lampert, M.D., Rapid City

Presently, the Board of Directors is comprised of eleven members; seven physicians and four lay people representing various businesses and industry.

Initially, South Dakota Blue Shield was funded through loans made to the Corporation by South Dakota physicians. Approximately \$15,000 was loaned to the Corporation by about 100 physicians.

Now, the operation of the Corporation is financed directly through premiums. These premiums are used to fund both benefit payments and administrative expenses. In 1979, the benefit payments were in excess of \$9 million—84.7% of the premium income.

One of the first official actions of the original corporate members was to elect John C. Foster as Secretary of the Corporation. Mr. Foster served as Executive Officer of South Dakota Blue Shield until 1963, at which time the duties were assumed by the current President, Richard C. Erickson.

The first Blue Shield contract in South Dakota was

sold in October, 1956. Since then more than 65,000 Blue Shield contracts have been sold, extending coverage to over 142,000 South Dakotans. The scope of benefits under these contracts has expanded significantly over the years, progressing from a limited indemnity type contract to a choice of contracts designed to meet the health care needs of most people.

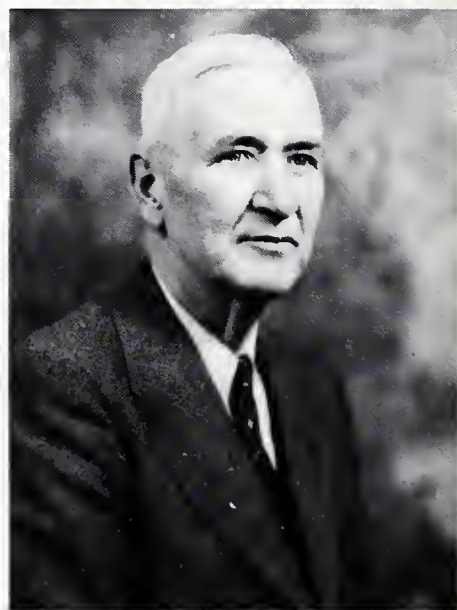
Over the years, South Dakota Blue Shield has served as fiscal administrator of many other programs such as Title 19 (Medicaid); CHAMPUS (Military Dependents Care); Old Age Assistance; and Social Security Medicare. In addition, we currently administer programs for Northwestern Bell Telephone Company, State and Federal employees. The past 25 years have brought about substantial growth in Blue Shield's operation; however, that growth has not overshadowed the original purpose of its existence—to offer South Dakotans the highest quality health care coverage and service at an affordable cost.

**YOUR CONTRIBUTION
IS NEEDED TO THE
SOUTH DAKOTA
MEDICAL SCHOOL
ENDOWMENT FUND**

First Graduate — USD School Of Medicine

Lyle Hare, M.D. Family Practitioner

Helen Jane Hare, M.D.*



When I was asked by the State Medical Association to write this story about my father, I thought first of those special qualities which made him the physician he was. This truly remarkable man loved people and accepted them as they were. Children could immediately sense this love and would come running to him and crawl into the haven of his lap. The elderly were understood and respected by him. His quiet, calm approach was comforting to those in need of help. When he entered a room, his patients could feel his compassion. It was a joy for me to have had this gentle doctor for my father.

In 1885 Lyle "Bunny" Hare was born in Cedar Rapids, Nebraska. When he was four years old the family moved to Hill City where his father operated a small cattle ranch and was in the newspaper business. "Bunny" attended country school, then later finished high school and college at Spearfish Normal School (now Black Hills State) where his training was financed with a football and track scholarship along with various odd jobs.

There were only two students in this first class when he entered Medical School at the University in 1906, again with a scholarship in football and track. By virtue of alphabetical listing he was Number One. The other student was named Iver Stoland.

To further pay his way, he delivered laundry and was houseboy at the Phi Delta Theta House. When he was asked how he could study with his jobs and athletic trips, he replied the faculty helped him a great deal. They really could not flunk half the class!! He really felt indebted to the University and his fraternity for the financial assistance without which he could not have studied medicine. During both years at USD he was unanimous choice for all conference full back and was mentioned by some sports writers in their selection of ALL AMERICAN TEAMS. As a track man, he gained considerable fame in weights and dash events at the Drake Relays.

After his graduation from USD, he completed his medical training at the University of Illinois Medical School in Chicago. When he was not in class he worked at a suburban hospital and interned at University Hospital.

He returned to Spearfish, South Dakota to enter private practice, be coach at Spearfish Normal School, as well as school physician and teacher of physiology. His football teams apparently were quite successful; however, an old timer said that when they played a four year school, my Dad was known to enter the fray along with his younger, lighter charges to even up the game. In two years his basketball teams lost only one game, this by only

*Dermatologist, Rapid City, SD; Clinical Professor, USD SM.

one point when a Spearfish player made a field goal—in the opponent’s basket.

Soon after moving to Spearfish he married his college sweetheart, Edna Stone, who died in the flu epidemic of 1918. In 1925 he was remarried. My wonderful stepmother, Hazel Beckman, also preceded him in death in 1972. His medical career in Spearfish spanned over fifty years. After two years, however, this active practice forced him to give up his coaching career, but he continued as school physician and teacher. For all those years he was also Homestake Physician.

He interrupted this practice to volunteer for duty in World War I. As a First Lieutenant in the Army Medical Corps he was assigned to a surgical section of a base hospital in France. In recent years I learned that he left for overseas immediately following my mother’s funeral. Until an uncle and aunt came for me, I was cared for by neighbors. I remember his telling me that due to problems with mail service, he had no word from home for two months after his arrival in France.

In those early years of his medical practice he was really a horse and buggy doctor. Sometimes he rode horseback. Often he was taken to remote areas by wagon. In winter he went by sleigh. I remember too the times when he would get stuck in gumbo for hours, or nearly froze to death in a stalled car in below zero weather. There were emergency calls made in a small plane with a “bush” pilot. If there was good pasture, that was where they landed; otherwise they came down where they could. After several close calls, my Dad no longer was very enthusiastic about this mode of transportation.

There was no hospital in Spearfish. His patients were hospitalized either in Deadwood or Belle Fourche which made many difficult drives on icy roads necessary. He was always on call. Patients occasionally came to our house nights and weekends. They also knew he would make house calls, some of them far out in the country. One time he performed an emergency operation on a woman with a ruptured tubal pregnancy. A kerosene lamp was the only illumination. Ether had to be used as an anesthetic. It must have been a frightening experience, but the woman’s life was saved.

A business man he was not. Monthly bills went out only from time to time as he got around to them. Most people came to the office to find out what they owed. Often they paid only what they thought they could. One day a man appeared wanting to pay for his own delivery as he knew the bill had never been paid. I cannot help but think what a problem he would have had with Medicaid, Medicare and the multiple insurance forms we have today. One could only speculate what his usual and customary fees

would have been. During the terrible years of the Great Depression we were well supplied with milk, cream, eggs, chickens and garden vegetables. There was no Medicaid in those bleak days, and for those who could not afford medical care my Dad made no charge.

How did he ever find the time, yet he felt a deep obligation to serve his community and state as well as his profession. During those hectic years he served as Mayor of Spearfish for two terms. He was an active member of the American Legion, serving as Commander and on the State Board, served as member and president of the governor’s Health Advisory Board, as a member and president of the State Board of Health, as a member and president of the State Board of Medical Examiners, as a member and president of the State Soldier’s Home Board, as a member and president of the District Medical Society, and during World War II was a member of the Lawrence County Selective Service Board. While he was attending school he was an active member of Phi Delta Theta and Nu Sigma Nu Medical Fraternity. He also was a 60 year member of Knights Templar and belonged for a time to the Naja Shrine Club.

A humble, unassuming man, he was quite overwhelmed by the many honors bestowed on him. For two successive years he was selected “General Practitioner of the Year” as well as being chosen by the American Medical Association as “first runnerup” for the same honor. The University of South Dakota presented him the honorary LLD in 1949 and the Distinguished Service Award in 1958; at the same time the Lyle Hare Medical Scholarship Award was founded. Black Hills State honored him by naming its fine new football field and all season track for him. Twelve years later the Lyle Hare Memorial Stadium was dedicated to him. In 1973 he was awarded the Presidential Award by the college. An athletic scholarship is awarded each year by the Purple and Gold Foundation of Black Hills State as a tribute to him. Finally, he was elected to the South Dakota Athletic Hall of Fame.

Lyle “Bunny” Hare died October 31, 1975, just a few weeks prior to his 91st birthday, after a long, debilitating illness. How pleased he would have been to have participated in our new four year medical school. What an inspiration he would have been to our students!

In preparing this biography I reviewed the material gathered at the time he was nominated for the General Practitioner of the Year Award. The testimonials to his character all stressed his devotion, compassion for all, and the respect and affection he had earned from his peers. That he was a fine physician I know from personal observation. His love and devotion to my mother, to me, and to my family are most cherished memories.

History Of The South Dakota State Medical Association Auxiliary 1910-1981

Muriel M. Reding*

The South Dakota State Medical Association Auxiliary is the **oldest, continuous Auxiliary in the nation!** There are complete records on file to substantiate this claim.

In September 1910 the South Dakota State Medical Association held its 29th annual meeting in Hot Springs. A group of physicians' wives, who had accompanied their husbands, called a meeting of their own. On September 29 these enthusiastic wives organized the Ladies Auxiliary to the South Dakota State Medical Association to "... bring the wives of physicians together in a spirit of good fellowship". Mrs. R. D. (Mattie) Jennings was elected first president. Dues were \$1.00. The fragile, tattered notebook with the minutes, a brief constitution and the names of the 18 Charter Members is preserved in the Auxiliary Archives file at the Medical Association office in Sioux Falls.

The Auxiliary continued to meet annually when the Medical Association held their convention. They planned programs and added new members. Later they divided into districts to match those of the Medical Association. In 1914 a committee ordered 300 pins for a membership of 50. Those ladies were optimistic . . . that number was not reached until 1953.

The Woman's Auxiliary to the American Medical Association was organized in 1922 in St. Louis during the annual meeting of the American Medical Association. The South Dakota Auxiliary, already 12 years old, became one of the first constituents of the national organization. South Dakota sent its first delegate to the national convention in 1924.

BENEVOLENT FUND BECOMES STUDENT LOAN FUND

A Benevolent Fund was established in 1938 to

assist indigent doctors and/or their families as soon as the \$5,000 goal was reached. The fund was dependent on donations from the districts and individuals so growth was slow. In 1947 the Auxiliary voted to add \$1.00 to the dues and asked the Medical Association to do the same. The goal was surpassed in 1954; then there was no need for its original purpose. A loan fund for needy medical students was discussed, the constitution amended and a joint committee (two from the Auxiliary and two from the Medical Association) was appointed to formulate plans for such a fund. The first \$1,200 loan was made in 1956. During the next 10 years 27 loans were made totaling \$32,000. Most of these loans have been repaid with interest. During the late sixties other sources for loans and grants became available to medical students, so little use was made of the fund.

In 1969 both the Auxiliary and the Medical Association voted to extend the use of this fund to include students in the allied health field. Again a joint committee adjusted the qualifications and changed the name to **Health Career Loan Fund**. Up to ten loans, not to exceed \$500 each, could be made annually to students attending South Dakota schools. Nearly 100 students have received aid from this fund to date. However, in 1981, due to complicated government regulations on such funds, only the interest from the principal of \$31,987.02 will be used in the form of grants . . . perhaps two of about \$500 this year.

AMA-ERF FOR THE MEDICAL SCHOOL

One of the highest priority projects of the South Dakota Auxiliary since the fifties has been AMA-ERF. An amazing total of \$124,111.74 has been raised by the Auxiliary during this time. Almost all money has been designated for the South Dakota School of Medicine. It is given to the Dean as an unrestricted fund although the Auxiliary does ask for an itemized statement annually.

*Historian and Past President, South Dakota State Medical Association Auxiliary, Marion, SD.

The American Medical Association Education and Research Foundation (AMA-ERF) was established by the AMA in 1951 and the Auxiliary and its constituents were asked for support. The purpose of the foundation was to provide financial assistance to medical schools (unrestricted funds available to Deans) and through the Student Loan Guarantee Fund to needy medical students, interns and residents.

South Dakota raised thousands of dollars in almost every conceivable way from selling Christmas Cards and watches to auctioning "donated" vacations to an ethnic dinner for six! Every type "benefit" has been used to raise dollars for AMA-ERF. The special AMA-ERF Auction during the state convention the last few years has brought hundreds of extra dollars because of the enthusiasm and generosity of the members of the Medical Association as well as the Auxiliary members.

SODAK MEDICAL AUXILIARY NEWS

The first mimeographed newsletter was sent out in 1950 by president, Muriel Reding and her officers, in a effort to bring the Auxiliary story to every doctor's wife in the state. It was to encourage membership and participation in Auxiliary projects and health oriented community programs. The newsletter proved successful and was continued . . . four issues a year. In the fall of 1953 the first printed newsletter was published, with the cost being underwritten by the Medical Association. The format had a sketch of Mount Rushmore in the left hand corner and the new name—**So Dak Medical Auxiliary News**.

The thick file of nearly 100 issues of the newsletter has become a chronicle that documents every important Auxiliary activity and is used as a reliable reference for Auxiliary leaders. It is truly a comprehensive history of the Auxiliary since 1950. The newsletter is sent to national officers and exchanged with other editors. It has been given national recognition and excerpts have been used by other editors and the national auxiliary magazine.

Muriel Reding continued as editor for 25 years and was honored by the Auxiliary in 1975 "... for her extraordinary service as editor"

VIRGINIA STOLTZ BECOMES NATIONAL PRESIDENT

When Mrs. C. Rodney Stoltz of Watertown was installed as president of the Woman's Auxiliary to the American Medical Association in June 1963, it had to be one of the proudest moments in the history of the South Dakota Auxiliary. Virginia had been recognized as a national leader during the ten years she served on the Board of Directors and as a mem-

ber of the Speakers' Bureau.

The State Medical Association and Auxiliary hosted a lovely reception to honor Virginia. More than 300 invited guests . . . officers of the Auxiliary and the AMA, delegates, state officers and friends came to congratulate Virginia and meet her family. The Medical Association presented her with a sterling silver coffee service and the Auxiliary added a matching silver compote.

Other South Dakota Auxilians who served on the national level as regional chairmen include: Muriel Reding (1952-1962), Dottie McIntosh (1974-76), Georgianna Bell (1978-80), Mrs. T. J. Billion, Sr. (1936), Mrs. J. R. Westaby (1938), Gladys Wold (1953) and Sue Volin (1954).

The Auxiliary celebrated its 50th Anniversary at the convention in 1960 in Aberdeen. The elaborate decorations, clever programs and favors all carried out the Golden Anniversary theme. Highlights of the first 50 years were noted and the history was printed in the newsletter. A handsome new gavel was presented to the Auxiliary by the president, Jane Brogdon, to commemorate the occasion. Special guests were the Golden Belles, wives of the Doctor's Fifty-Year Club, and all long-time Auxiliary members. There were two national guests.

Through the years the Auxiliary has continued to work for better health legislation, to provide programs and materials on health subjects such as venereal disease, child abuse and the battered child, alcoholism, nutrition, immunization, drug abuse, physical fitness, health care cost containment and the like. Members have supported health-related charitable endeavors and sponsored vision and hearing clinics in their communities. Membership has reached 386 with three honorary members. There are 10 organized districts and members-at-large in the other two. In 1977, the Auxiliary name was changed to South Dakota State Medical Association Auxiliary in order to extend membership to husbands of women physicians.

The Auxiliary has enjoyed full cooperation from the Medical Association and is most grateful for the financial assistance of the newsletter and the support for AMA-ERF projects. In 1966 the Medical Association presented its highest honor, the Distinguished Service Award, to Virginia Stoltz.


This is only a general resume of the activities of the South Dakota Auxiliary. A complete history will be published in 1985 when the Diamond Anniversary will be celebrated.

Congratulations to the South Dakota State Medical Association for 100 Years of Service to the People of South Dakota!



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is no laughing matter.**

The first prescription for the first days of acute pain Empirin® \bar{c} Codeine #3


Each tablet contains: aspirin, 325 mg; plus codeine phosphate, 30 mg, (Warning — may be habit-forming). 

For the millions of patients who need the potency of aspirin and codeine for their acute pain.

The pain of fractures, strains, sprains, burns and wounds is at its peak during the first three to four days following trauma. The potent action of Empirin \bar{c} Codeine begins to work within 15 minutes of oral administration, an important advantage during this acute pain period. Empirin \bar{c} Codeine has unique bi-level action to attack pain at two critical points: peripherally at the site of injury and centrally at the site of pain awareness.

For the most effective dosage in treating acute pain, begin with ... two tablets of Empirin \bar{c} Codeine #2 or #3, every four hours. Titrate downward as pain subsides.

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DESCRIPTION: Each tablet contains aspirin (acetylsalicylic acid) 325 mg plus codeine phosphate in one of the following strengths: No. 2 — 15 mg, No. 3 — 30 mg, and No. 4 — 60 mg. (Warning — may be habit-forming.) 

CONTRAINDICATIONS: Hypersensitivity to aspirin or codeine.

WARNINGS:

Drug dependence: Empirin with Codeine can produce drug dependence of the morphine type and, therefore, has the potential for being abused. Psychic dependence, physical dependence, and tolerance may develop upon repeated administration of this drug and it should be prescribed and administered with the same degree of caution appropriate to the use of other oral, narcotic-containing medications. Like other narcotic-containing medications, the drug is subject to the Federal Controlled Substances Act.

Use in ambulatory patients: Empirin with Codeine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. The patient using this drug should be cautioned accordingly.

Interaction with other central nervous system (CNS) depressants: Patients receiving other narcotic analgesics, general anesthetics, phenothiazines, other tranquilizers, sedative-hypnotics, or other CNS depressants (including alcohol) concomitantly with Empirin with Codeine may exhibit an additive CNS depression. When such combined therapy is contemplated, the dose of one or both agents should be reduced.

Use in pregnancy: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. Therefore, Empirin with Codeine should not be used in pregnant women unless, in the judgment of the physician, the potential benefits outweigh the possible hazards.

PRECAUTIONS:

Head injury and increased intracranial pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a pre-existing increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.

Acute abdominal conditions: The administration of Empirin with Codeine or other narcotics may obscure the diagnosis or clinical course in patients with acute abdominal conditions.

Allergic: Precautions should be taken in administering salicylates to persons with known allergies: patients with nasal polyps are more likely to be hypersensitive to aspirin.

Special risk patients: Empirin with Codeine should be given with caution to certain patients such as the elderly or debilitated, and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture, peptic ulcer, or coagulation disorders.

ADVERSE REACTIONS: The most frequently observed adverse reactions to codeine include light-headedness, dizziness, sedation, nausea and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include euphoria, dysphoria, constipation, and pruritus.

The most frequently observed reactions to aspirin include headache, vertigo, ringing in the ears, mental confusion, drowsiness, sweating, thirst, nausea, and vomiting. Occasional patients experience gastric irritation and bleeding with aspirin. Some patients are unable to take salicylates without developing nausea and vomiting. Hypersensitivity may be manifested by a skin rash or even an anaphylactic reaction. With these exceptions, most of the side effects occur after repeated administration of large doses.

DOSE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient. It may occasionally be necessary to exceed the usual dosage recommended below in cases of more severe pain or in those patients who have become tolerant to the analgesic effect of narcotics. Empirin with Codeine is given orally. The usual adult dose for Empirin with Codeine No. 2 and No. 3 is one or two tablets every four hours as required. The usual adult dose for Empirin with Codeine No. 4 is one tablet every four hours as required.

DRUG INTERACTIONS: The CNS depressant effects of Empirin with Codeine may be additive with that of other CNS depressants.

WARNINGS:



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Medical Highlights In South Dakota—The First 100 Years Of The South Dakota State Medical Association

- 1861 President Abraham Lincoln appointed Dr. William Jayne, Springfield, Illinois, first governor of the Territory of Dakota.
- 1869 The first legislative act restricting those practicing medicine in Dakota Territory was passed.
- 1882 Dakota physicians met in Milbank at the Grand Central Hotel on June 3, 1882, to form a medical society for the Dakota Territory. The official name was the Dakota Medical Association but it was called the Dakota Medical Society until 1906.
- 1885 The territorial legislature, at the urging of the medical society, adopted statutes creating territorial and county boards of health to protect the health of persons and animals.
- 1886 The Dakota Medical Brief was established as the Dakota Medical Society's official publication. The Society seal was developed and the Society was authorized to print certificates of membership.
- 1887 The Society received information on the first "quackery" practice of medicine in the territory.
- 1890 Because South Dakota attained statehood in 1889, the name of the Society was changed to South Dakota State Medical Society.
- 1891 The South Dakota State Medical Society was incorporated on May 29, 1891.
- 1903 The Society was reorganized to include nine district societies.
- 1903 The legislature at the urging of the State Society established the State Board of Medical Examiners and adopted legislation regulating the practice of medicine.

(continued)

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The #1 premoistened
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external relief and gentle
cleansing of fecal residue
that can irritate
tender anorectal tissue.

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*Data on file Parke-Davis Marketing Research Dept.
**Based on total prescriptions filled for hemorrhoidal preparations during the first three quarters of 1980. The National Prescription Audit, IMS America Ltd., September 1980.

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Hemorrhoids and other anorectal uses—TUCKS extra-soft cloth pads allow for the gentlest possible application to tender, inflamed, hemorrhoidal tissue. TUCKS are effective cleansing pads for everyday personal hygiene. Used on outer rectal areas, they remove residue that can bring on more irritation. Pads are premoistened with 50% witchhazel, 10% glycerin USP and de-ionized purified water USP which acts as a cooling, soothing lotion to help comfort sensitive anorectal tissue.

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ANUSOL-HC® SUPPOSITORIES

Hemorrhoidal Suppositories with Hydrocortisone Acetate

ANUSOL-HC® CREAM

Rectal Cream with Hydrocortisone Acetate

Caution: Federal law prohibits dispensing without prescription.

Description: Each Anusol-HC Suppository contains hydrocortisone acetate, 10.0 mg; bismuth subgallate, 2.25%; bismuth resorcin compound, 1.75%; benzyl benzoate, 1.2%; Peruvian balsam, 1.8%; zinc oxide, 11.0%; also contains the following inactive ingredients: dibasic calcium phosphate, and certified coloring in a hydrogenated vegetable oil base.

Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg; bismuth subgallate, 22.5 mg; bismuth resorcin compound, 17.5 mg; benzyl benzoate, 12.0 mg; Peruvian balsam, 18.0 mg; zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water.

Anusol-HC Suppositories and Anusol-HC Cream help to relieve pain, itching and discomfort arising from irritated anorectal tissues. These preparations have a soothing, lubricant action on mucous membranes, and the antiinflammatory action of hydrocortisone acetate in Anusol-HC helps to reduce hyperemia and swelling.

The hydrocortisone acetate in Anusol-HC is primarily effective because of its antiinflammatory, antipruritic and vasoconstrictive actions.

Indications and Usage: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain, itching and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas, pruritus ani and relief of local pain and discomfort following anorectal surgery.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

Contraindications: Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

Precautions: General: Symptomatic relief should not delay definitive diagnoses or treatment.

Prolonged or excessive use of corticosteroids might produce systemic effects.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Anusol-HC is not for ophthalmic use.

Pregnancy

See "WARNINGS"

Pediatric Use

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

Dosage and Administration: Anusol-HC Suppositories—

Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain comfort with regular Anusol Suppositories.

Anusol-HC Cream—Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

How Supplied: Anusol-HC Suppositories—boxes of 12

(N 0071-1089-07) and boxes of 24 (N 0071-1089-13) in silver foil strips with Anusol-HC printed in black.

Anusol-HC Cream—one-ounce tube (N 0071-3090-13) with plastic applicator.

Store between 59°-86°F (15°-30°C).

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PARKE-DAVIS

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- 1906 The Northwestern Lancet became the official publication of the Society. Minnesota and North Dakota shared this publication with South Dakota.
- 1906 The South Dakota Legislature approved funding for the two year School of Medicine at the University of South Dakota and accepted its first class (two students).
- 1908 The Society appointed itself as guardian of medical legislation and instituted the use of lobbyists in Pierre during legislative sessions.
- 1909 Legislation was passed creating a State Laboratory in Vermillion. This bill was promoted by the Medical Society.
- 1910 The State Medical Association Auxiliary was formed. This is the oldest continuous medical auxiliary in the United States.
- 1912 The Tenth District (Rosebud) was formed.
- 1913 The Medical Society drafted legislation creating a State Board of Health; and the law passed with minor amendments.
- 1916 The Association established a Committee on Medical Defense. This committee established an insurance program to protect its members from law suits arising from their practice.
- 1928 South Dakota joined the Regional State Medical Conference which included North and South Dakota, Wisconsin, Minnesota and Iowa. This has since evolved into the North Central Medical Conference.
- 1928 The first grant awarded by the Medical Association was a \$25 award to a University of South Dakota student showing proficiency in Latin, preferably Virgil.
- 1939 The legislature passed a bill establishing the Basic Science Board. This Board was sunsetted in 1979.
- 1939 The Association and Auxiliary established a benevolent fund to provide financial assistance to South Dakota physicians in need. This has since been revised to a loan program for students in medical school, a loan program for allied health professionals and now a grant program for allied health persons.
- 1945 The State Medical Association introduced legislation providing that the State Board of Health should license hospitals; this law was passed.
- 1946 The Association hired its first full time administrator, John Foster, and established an office at 300 First National Bank Building, Sioux Falls.

(continued)

- 1946 The Association first proposed to the legislature expansion of the two year basic science school to a four year degree granting medical school. This legislation failed.
- 1947 The South Dakota State Medical Association formed the South Dakota Injury-Illness Expense Plan, a prepaid surgical-medical, nursing and hospital indemnity plan. This program was underwritten by private insurance carriers and governed jointly by the State Medical Association and the insurance carriers. By 1948 there were 4,353 individuals insured under this plan.
- 1948 The South Dakota Journal of Medicine and Pharmacy became the official publication of the Association.
- 1949 The South Dakota Medical School Endowment Association was established to collect money to assist the Medical School and provide student loans. This fund currently loans approximately \$20,000 annually to USDSM students in addition to providing other money for the school.
- 1950 The Association established a placement service to facilitate placement of rural physicians.
- 1956 The South Dakota State Medical Association and the North Dakota State Medical Association met jointly in Aberdeen to celebrate the SDSMA diamond jubilee. Two hundred five South Dakota physicians attended this meeting.
- 1956 Interns and residents were recognized as courtesy members of the South Dakota State Medical Association. As of 1981 they are eligible for voting privileges as associate members.
- 1956 South Dakota Blue Shield was established.
- 1957 Physicians throughout South Dakota participated in clinics providing Salk vaccine for polio immunizations.
- 1959 The State Medical Association reinstituted the Medical Defense Committee to evaluate proposed malpractice suits and make a determination on the validity of such suits.
- 1959 The State Medical Association purchased land on North Lake Avenue, Sioux Falls, for an executive office building.
- 1960 A Relative Value Study was adopted by the State Medical Association. Because of action taken by the Federal Trade Commission against other state Relative Values, the State Medical Association rescinded this Study in 1977.

(continued)

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- 1960 The Association moved into its new office building in Sioux Falls.
- 1960 Medicine became involved in government and candidate support by organizing the South Dakota Physicians Committee which became the South Dakota Political Action Committee (SoDaPAC) in 1963.
- 1962 South Dakota physicians promoted mass oral polio vaccine immunization clinics.
- 1964 Richard C. Erickson was appointed executive secretary of South Dakota State Medical Association and South Dakota Blue Shield. John Foster became executive director of Arizona Blue Cross-Blue Shield.
- 1965 The pharmacy section of the Journal was discontinued and the publication became the South Dakota Journal of Medicine.
- 1966 The Medicare program was implemented.
- 1967 Comprehensive Health Planning Program was implemented in South Dakota.
- 1967 An addition to the executive office was completed. Two more additions were completed in 1968 and 1970.
- 1971 Membership in the State Medical Association was extended to doctors of osteopathy.
- 1973 Robert D. Johnson assumed the duties of executive secretary for the State Medical Association. Richard Erickson remained as executive director for South Dakota Blue Shield.
- 1974 The Foundation for Medical Care which operates the PSRO program in South Dakota was established.
- 1974 The State Medical Association moved to new headquarters at 608 West Avenue, North, Sioux Falls, South Dakota.
- 1974 The University of South Dakota School of Medicine was expanded to a four year degree granting school. The first graduating class was in 1977.
- 1975 South Dakota, as well as the entire United States, was involved in a professional liability insurance crisis. Numerous bills were introduced and passed by the legislature in the next few years to help alleviate this problem.
- 1976 Physicians in South Dakota participated in the swine flu immunization program.
- 1978 The Voluntary Effort, a joint program of the Hospital Association and the Medical Association to monitor and control health care costs, was established.
- 1981 The SDSMA celebrates its centennial year.

REMEMBER THE ENDOWMENT ASSOCIATION!

*Name the South Dakota Medical School Endowment
Association as the beneficiary of a life
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Your contributions
and assistance will
benefit the USD School
of Medicine and its
students.

South Dakota Medical School Endowment Association
608 West Avenue, North
Sioux Falls, SD 57104

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Future Meetings

June

John Lawrence Interdisciplinary Symposium on the Physical and Biomedical Sciences, Sioux Falls, SD, June 3-4. Contact: George P. Scott, Dept. of Chemistry, Univ. of SD, Vermillion, SD 57069.

Physician Recruitment, Am. Hosp. Assoc. Hdqtrs., Chicago, Ill., June 11-12. Fee: \$200. Contact: Tom Atchison, Am. Hosp. Assoc., Box 96003, Chicago, IL 60690. Phone: (312) 280-6449.

Clinical Management of Coronary Disease and Exercise Testing, Sahara Hotel, Las Vegas, NV, June 12-14. 13 hrs. AAFP & AMA Category I credit. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

Intensive Course in Pediatric Nutrition, Univ. of Iowa, Iowa City, IA, June 15-19. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa Coll. of Med., Iowa City, IA 52242.

Emergency Medical Services: Responsibility and Accountability in the '80's, Copley Plaza Hotel, Boston, Mass., June 17-19. Fee: \$285. Contact: EMS And The Law, Am. Society of Law and Med., 520 Commonwealth Ave., #211, Boston, MA 02215. Phone: (617) 262-4990.

Arrhythmias and Cardiac Ischemia: Diagnosis and Management, Dutch Inn, Orlando, FL, June 26-28. 13 hrs. AAFP & AMA Category I credits. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

EKG Interpretation and Arrhythmia Management, Sheraton Anaheim, Anaheim, CA, June 26-28. 13 hrs. AAFP & AMA Category I credit. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

July

Clinical Management of Coronary Disease and Exercise Testing, The Abbey Resort, Lake Geneva, WI, July 17-19. 13 hrs. AAFP & AMA Category I credit. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

Cardiac Rehabilitation, Orlando Hyatt, Orlando, FL, July 24-25. 13 hrs. AAFP & AMA Category I credit. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

EKG Interpretation and Arrhythmia Management, Shanty Creek Hilton Resort, Bellaire, MI, July 31-Aug. 2. 13 hrs. AAFP & AMA Category I credit. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

August

Advances in Diagnostic Imaging, Hotel Scandinavia, Copenhagen, Denmark, Aug. 2-8. 30 hrs. AMA Category I credits. Contact: Dr. L. R. Muroff, Box 17241, Tampa, FL 33682. Phone: (813) 971-6000, ext. 297.

Great Debates in Otolaryngology, Washington Plaza Hotel, Seattle, WA, Aug. 6-8. 19.5 hrs. AAFP & AMA Category I credit. Fee: \$350. Contact: Registration, Cont. Med. Ed., SC-50, Univ. of Wash., Seattle, WA 98195. Phone: (206) 543-1050.

EKG Interpretation and Arrhythmias Management, Doubletree Inn, Monterey, CA, Aug. 7-8. 13 hrs. AAFP & AMA Category I credit. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

Arrhythmias and Cardiac Ischemia: Diagnosis & Management, Hyatt Regency, Montreal, Canada, Aug. 14-16. 13 hrs. AAFP & AMA Category I credits. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

The Sixth Annual Convention of the American College of International Physicians, Holiday Inn, Chicago, Ill., Aug. 20-23. 12 hrs. AMA Category I Credit. Contact: Am. College of International Phys., 3030 Lake Ave., Fort Wayne, IN 46805. Phone: (219) 424-7414.

Cardiac Rehabilitation, Sheraton Anaheim, Anaheim, CA, Aug. 21-22. 13 hrs. AAFP & AMA Category I credits. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

Recent Advances in Diabetes Management, Glacier Park Lodge, East Glacier, Mont., Aug. 29. 7-8 hrs. Category I credit. Contact: Stanlee Dull, Exec. Dir., Am. Diabetes Assoc., Box 2411, Great Falls, MT 59403. Phone: (406) 761-0908.

October

The Sixth Annual International Body Imaging Conference, Hyatt Regency Hotel, Maui, Hawaii, Oct. 10-18. 25 hrs. Category I credit. Fee: \$365. Contact: Conference Secretary, Sixth Annual Internat'l. Body Imaging Conf., West Park Hosp., Dept. of Radiology, 22141 Roscoe Blvd., Canoga Park, CA 91304.

SOUTH DAKOTA JOURNAL OF MEDICINE

Published Monthly by the S.D. State Medical Assn.

Volume XXXIV June 1981 Number 6



Clinicopathological Conference
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Family Physician Needs For South Dakota—1990

Table of Contents: page 3

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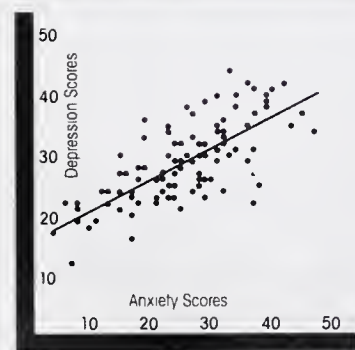
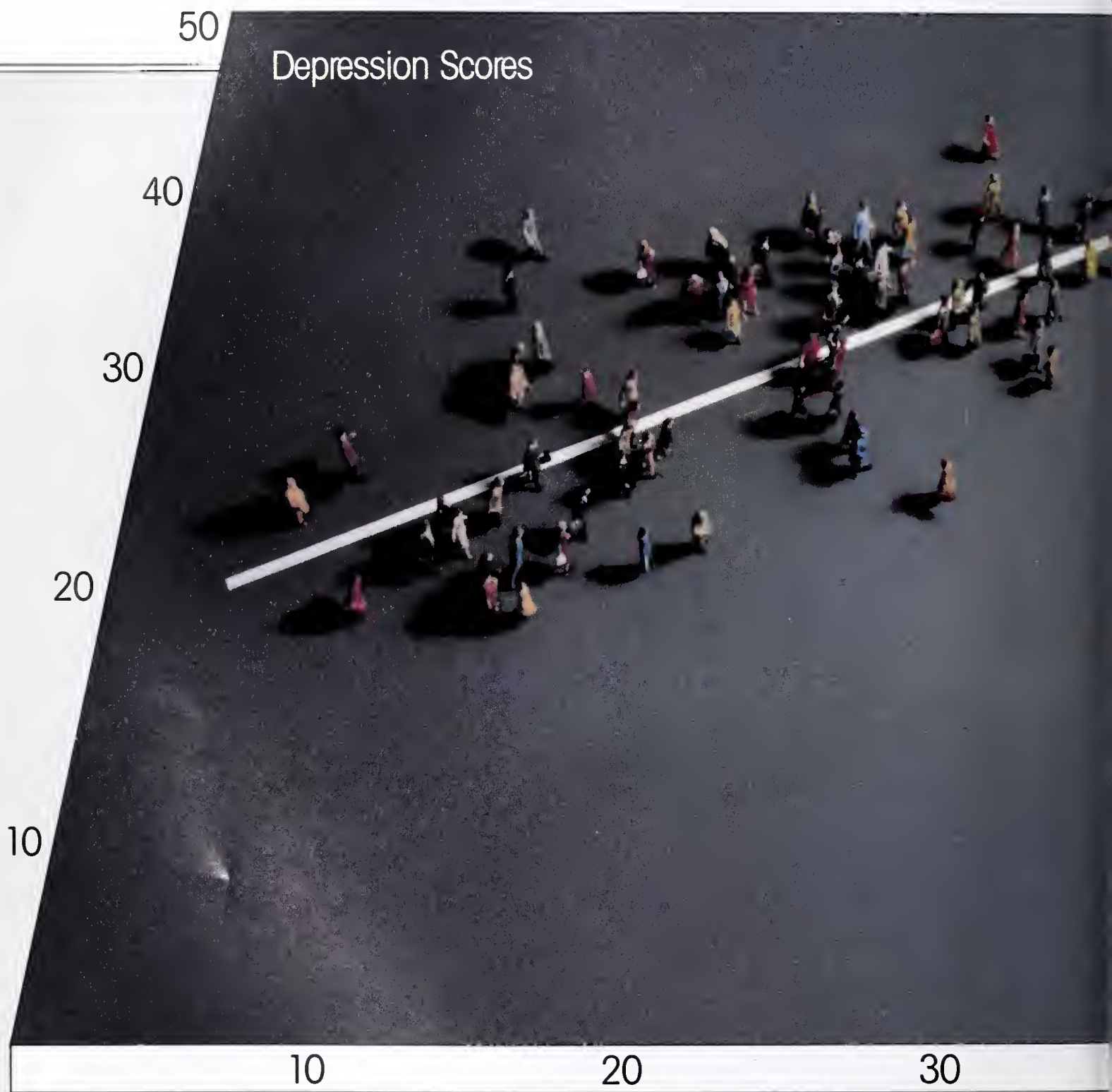
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FOR THE 7 OF 10 NONPSYCHOTIC



Clear correlation between anxiety and depression³

The above graph illustrates a relationship between anxiety and depression, indicating that patients seldom present with anxiety or depression alone, more often they have both in varying degrees. Data based on a sampling of 100 outpatients (64 male, 36 female) seen at a general psychiatric clinic.

³Adapted from Claghorn, J. The anxiety-depression syndrome. *Psychosomatics* 11:438-441, Sept-Oct 1970.

DEPRESSED PATIENTS WHO ARE ALSO ANXIOUS^{1,2}

Most depressed patients are also anxious. . .

Some authors estimate that 70% of all nonpsychotic patients with symptoms of depression have concomitant symptoms of anxiety.^{1,2} One author found a distinct correlation between anxiety and depression scores in 100 nonpsychotic outpatients administered the Minnesota Multiphasic Personality Inventory in a general psychiatric clinic.³ As depression scores increased, so did anxiety scores. No attempt was made to select patients other than to exclude psychotics.

but not psychotic

The logic of treating both components of anxious depression is clear. Antipsychotics, like the phenothiazines, however, carry a well-documented risk of tardive dyskinesia.⁴ Because of this, an APA Task Force recently recommended the judicious use of phenothiazines in cases other than chronic psychosis or the use of alternative treatments.

A better way to give relief

Limbitrol combines the specific anxiolytic action of Librium® (chlordiazepoxide HCl/Roche)—a benzodiazepine with a long history of safe use—with the antidepressant action of amitriptyline, a tricyclic of established clinical efficacy. In comparison to phenothiazines, Limbitrol and its components have rarely been associated with tardive dyskinesia or other extrapyramidal side effects. And in terms of rapid response and patient compliance, Limbitrol appears to be superior to amitriptyline alone. Controlled multiclinic studies showed Limbitrol relieved more symptoms more rapidly than did amitriptyline.⁵ Despite a higher incidence of drowsiness, the dropout rate due to side effects was lower with Limbitrol. (See adverse reactions section in summary of product information on next page. As with any CNS-acting agent, patients should be cautioned about driving or using dangerous machines while on therapy with Limbitrol.)

References: 1. Rickels K: Drug treatment of anxiety, in *Psychopharmacology in the Practice of Medicine*, ed. Jarvik ME. New York, Appleton-Century-Crofts, 1977, p. 316. 2. Schatzberg AF, Cole JO: Benzodiazepines in depressive disorders. *Arch Gen Psychiatry* 35:1359-1365, 1978. 3. Claghorn J: The anxiety-depression syndrome. *Psychosomatics* 11:438-441, 1970. 4. The Task Force on Late Neurological Effects of Antipsychotic Drugs: Tardive dyskinesia, summary of a task force report of the American Psychiatric Association. *Am J Psychiatry* 137:1163-1172, 1980. 5. Feighner JP *et al*: A placebo-controlled multicenter trial of Limbitrol versus its components (amitriptyline and chlordiazepoxide) in the symptomatic treatment of depressive illness. *Psychopharmacology* 61:217-225, 1979.

Anxiety Scores

50

In moderate depression and anxiety

Limbitrol® ®

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

Relief without a phenothiazine

Please see summary of product information on next page.

LIMBITROL® TABLETS Tranquilizer—Antidepressant

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of moderate to severe depression associated with moderate to severe anxiety

Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use, then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.

Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.) Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving).

Usage in Pregnancy: Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage, withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline, symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).

Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action at guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated; sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration at ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12.

In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.

Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.

The following list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.

Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.

Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.

Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.

Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.

Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.

Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

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Sixteen Year Old Caucasian Male With Rapidly Progressive Dyspnea And Dysphagia

Kari D. Olsen, M.D.*
Discussor

John F. Barlow, M.D.**
Editor

Case No. 871 271 3

This 16 year old Caucasian male entered the emergency room of Sioux Valley Hospital because of inspiratory stridor and dysphagia.

The patient had been seen multiple times in the emergency room for complaints of abdominal pain, vomiting, and swelling of the arms, heels, or face. The patient was diagnosed at the age of 3 as having hereditary angioedema. The patient's father died in his 20's reportedly of this condition. The patient's grandmother and one uncle had the disorder. He had been seen at the Mayo Clinic as recently as a year previous and given recommendations for treatment including chlorpromazine.

The patient awoke on the morning of admission with mild sore throat, which progressed until he was having minor difficulty swallowing and breathing. He had returned home and was resting but when the telephone rang, he arose abruptly and noted that he was having marked difficulty breathing and could not speak. He summoned an ambulance although unable to talk. Because his telephone number was traced, he was brought to the Sioux Valley emergency room. He was noted to have severe inspiratory stridor. He was given epinephrine 1:10,000 dilution, 0.5 mgs. and 500 mgs. of methylprednisolone (solumedrol) as well as 50 mgs diphenhydramine (benadryl). There was some resolution of the stridor. He received another 0.3 mgs of epinephrine and the stridor faded completely.

The patient had no other known hospitalizations except for the multiple emergency room visits. He had had no surgery or other serious illnesses. A review of systems was unremarkable, except for the attacks of abdominal pain which would last

24 hours and occur once or twice a month. These were precipitated mostly by fatigue or stress. There was marked vomiting during these attacks but they resolved spontaneously with the help of diphenhydramine (benadryl).

PHYSICAL EXAMINATION: Temperature 98°F, pulse 85/min. and regular, respirations 20/min. and regular. Blood pressure 110 systolic and 70 diastolic. The patient was in severe respiratory distress and had difficulty swallowing saliva. There was no evidence of facial edema. Examination of the head and neck was unremarkable. The lungs were clear to auscultation and percussion after the patient was able to move air. The heart was not enlarged. There was a soft grade I (six grades) systolic heart murmur, loudest at the left costal margin at the second intercostal space. Examination of the abdomen showed no tenderness, organs, or masses. The genitalia were normal male. The extremities showed some diffuse swelling on the left forearm and both heels and mild back tenderness in the lumbar muscle groups. Neurologic examination was within normal limits.

LABORATORY DATA: Urinalysis-clear yellow, specific gravity 1.016, negative for protein, glucose, ketone bodies, bile, hemoglobin; sediment negative. Hemoglobin 16.0 gm/dl, Hct 45 vol/dl, normal red cell indices, total leukocyte count 13,900/mm³ (13.9 x 10⁹/L) with 77% segmented neutrophils, 11% neutrophilic bands, 12% normal lymphocytes. The red cells were normochromic normocytic and the platelets normal in number and morphology on the smear. Soft tissue lateral view of the neck showed narrowing of the airway. An electrocardiogram was interpreted as sinus rhythm.

The patient was discharged after observation for a few days.

DR. OLSEN: Hereditary angioedema (HAE) is a familial autosomal dominant disorder which is characterized by recurrent episodes of edema of the

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skin, respiratory tract, and gastrointestinal tract. It is due to a deficiency of an inhibitor protein of the activated first component of the complement system.

The disease is characterized by subcutaneous swelling of the skin, most commonly over the extremities and face. The edema is distinctly non-inflammatory in appearance. HAE is not a form of urticaria. It may first develop with areas of serpiginous erythema and mottling, but most commonly the skin manifestations are non-erythematous, non-pitting, non-pruritic, and non-painful. Occasionally pain may be present when the swelling is so severe that the skin becomes distended and the edema may pit as the attack subsides. Patients may be unable to flex the fingers if the hands are markedly swollen. Some patients may describe a peculiar sensation or tingling over the skin prior to development of angioedema. In general, although bothersome to the patients, the skin manifestations are much less serious than involvement elsewhere.

Involvement of the gastrointestinal tract produces extreme abdominal pain with vomiting. The pain is diffuse and colicky. Some patients may experience a watery diarrhea especially as the attack subsides. The clinical picture may mimic intestinal obstruction and many patients have been subjected to multiple exploratory laparotomies. Indeed, the edema may become so pronounced that the bowel becomes transiently obstructed. The most common area of involvement is in the jejunum. In addition, the edema of the gut wall may become so great that hypovolemia and shock ensue. On examination, the abdomen may be tender but signs of peritoneal irritation are not present. Abdominal films are non-specific and show evidence of edema of the gut wall. Occasionally a slight fever will be noted. The white blood count may be slightly elevated secondary to hemoconcentration, but there is no left shift in the neutrophilic series. The attack is gradual in onset and resolves within 48-72 hours.

Episodes of edema of the respiratory tract present the most dangerous threat to the patient. Serious airway obstruction will occur in two-thirds of patients and laryngeal edema is the cause of death in 25% of cases. Although respiratory obstruction usually develops slowly, some affected kindreds are prone to sudden death from laryngeal edema with pulmonary edema. Fortunately, many patients experience a voice change or hoarseness⁵ followed by pooling of secretions and dysphagia prior to total airway obstruction. When a patient presents with pharyngeal involvement, hospitalization is mandatory for evaluation of airway obstruction. Many patients will require tracheostomy, although early in an attack, patients may be managed by nasotracheal

or endotracheal intubation. These tubes are left in place for 24-48 hours.

In rare instances angioedema may develop in the pleura causing pleuritic chest pain and cough. Pleural effusions may be noted which resolve spontaneously. There have also been reports of angioedema involving the brain producing seizures and hemiparesis.

Involvement of the skin, gastrointestinal tract, or airway may occur together or independently. When only one organ system is affected in an attack, the diagnosis of HAE may be particularly elusive. In fact, in one series, the average time between onset of symptoms and diagnosis was 20 years.

The age of onset of symptoms is extremely variable. Attacks may begin in early childhood but are infrequent in this age group. The frequency increases in adolescence and subsides after the fifth decade. Some women notice an increase in frequency of attacks during menses but a few report improvement with menopause. Birth control pills may aggravate the disorder. Pregnancy seems to have a dramatic effect in that most series report a marked decline in attacks especially in the second and third trimesters. Interestingly, in several series there were no attacks of angioedema associated with delivery. This is an unexpected finding since trauma is thought to be one of the major initiators in an attack of HAE.

50% or more of patients report trauma as a precipitating event to an episode. The trauma can be quite minor in nature and activities such as typing, lawn mowing, and hammering have been incriminated. Any surgical or dental procedure may trigger an event and laryngeal edema should be anticipated with oropharyngeal manipulation. An atopic history is notably absent in most patients. There is no evidence at present to indicate an allergic component to HAE. Many patients report emotional stress or fatigue as important factors in provoking an attack.

Although first described in 1843 by Graves, the pathophysiology of HAE is only now beginning to be defined. The defect in this disorder is a deficiency or a functional lack of the inhibitor (C1-INH) of the first component (C1) of the complement cascade.

The complement system consists of a series of proteins which successively react with antigen-antibody complexes, macromolecular aggregates, or sensitized cells. Its function has been best described in relation to its role in immune hemolysis. Nine components of complement are known. The classical pathway of complement is activated via antigen-antibody aggregates. The activated first component of complement ($C1$) is an esterase and acts on its substrate C4 and C2 to generate an activated complex $C42$. $C42$ then acts on C3 and in a cleavage reaction produces 2 fragments C3b and C3a. Once

C3 is cleaved the remainder of the components C5-C9 are induced to function (See Fig. 1).

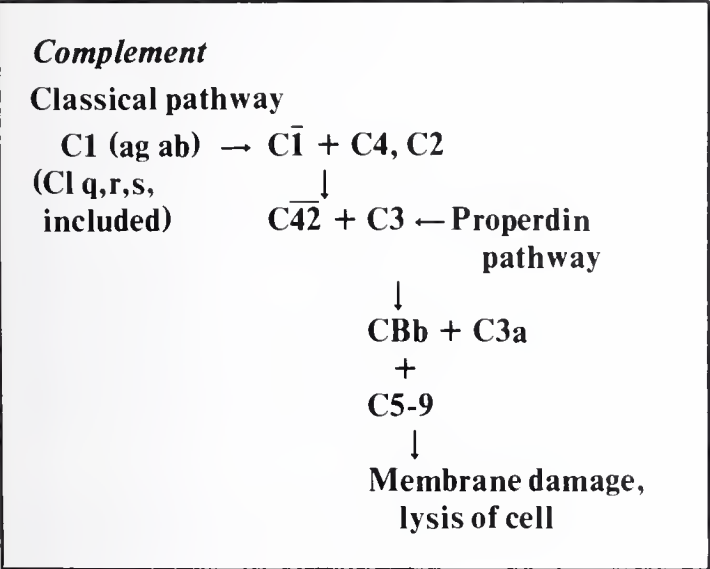


Figure 1
Classical and properdin pathways are briefly outlined. ag-ab = antigen-antibody, Bar (-) indicates activation of factor.

C1 is a macromolecular complex composed of 3 subunits C1q, C1r, and C1s which are bound together by Ca ++. C1q is the subunit which binds to the Fc portion of the antibody. This C1q-Ab complex activates C1r which is a proenzyme and converts it to an active enzyme. C1r in turn activates C1s which acts as an esterase. The activated first component of complement then, is an esterolytic enzyme (C1 esterase) (See Fig. 1).

The complement cascade can also be activated by an alternate pathway, the properdin pathway. It activates the cascade at the C3 level. Polysaccharides, endotoxin, and cobra venom factor have been identified as mediators in this pathway.

C1 inhibitor (C1-INH) is a protein that regulates the activity of C1. It has a molecular weight of 90,000 and is probably synthesized in the liver. It is an alpha-2 globulin and an acute phase reactant. C1-INH also has functions in conjunction with other serum proteins as an inhibitor in other enzyme pathways. C1-INH can inhibit kallikrein, plasmin, activated Hageman factor (Factor XIIa) and activated plasma thromboplastin antecedent (Factor XIa).

There are two forms of HAE. In 85% of cases the disorder is due to low levels of antigenically and functionally normal C1-INH. In the remaining 15% of cases, there is a normal to increased level of antigenically normal protein which has abnormal function.

C1-INH combines with the C1s subunit to block its esterase activity. It has also been shown to inhibit the C1r subunit. In the absence of C1-INH, the first component of complement is activated spontane-

ously. C1 may also be activated by trypsin, plasmin, thrombin, and kallikrein. This explains why trauma is an important factor in triggering an attack. Trauma activates Hageman factor which in turn activates clotting, kinin generation, and plasmin formation. There is no evidence that antigen-antibody interaction causes complement activation in HAE.

During an attack of angioedema, the level of activated C1s increases and its substrates, C4 and C2, are quickly depleted so that C4 may be undetectable in the blood. For reasons which are not completely known, the uncontrolled activation of complement does not affect the entire cascade. C3 and the later components of complement are not involved and their blood levels remain normal. Since C4 levels are decreased in HAE, measurement of C4 by radial immunodiffusion assay provides a good screening test. A normal or high concentration of C4 excludes the diagnosis (See Fig. 2).

Laboratory Evaluation of HAE

Screening test: C4 by radial immunodiffusion assay (RID)

If C4 decreased or normal then measure C1-INH

a. If C4 decreased and C1-INH decreased—common form of HAE

b. If C4 decreased and C1-INH normal or increased—variant form of HAE

To confirm variant form requires functional C1-INH assay

C4 = fourth component of complement; C1-INH = first component of complement inhibitor;

HAE = hereditary angioedema.

Figure 2

The pathologic changes seen in HAE are those secondary to edema. This is thought to be due to increased vascular permeability at the post capillary venule. The exact mechanism for edema formation is unclear. There are probably multiple mediators involved. Some evidence points to increased levels of a polypeptide with kinin activity which is thought to be generated by C2. This so called C2 kinin produces increased capillary permeability. Increased levels of histamine in serum and urine suggests a possible role for it as a mediator. However, the peak levels of histamine are not noted until the attack is subsiding indicating that the increased levels may be a secondary finding. In addition, the administration of antihistamines during an attack seems to have little clinical effect.

In a suspected case of HAE, a good screening test is the radial immunodiffusion assay for C4. If a normal or low C4 level is obtained, then a C1-INH level should be quantitated by radial immunodiffusion assay. If both the C4 and C1-INH levels are low, the common form of HAE is the most likely diagnosis (See Fig. 2).

If the C4 is low and the C1-INH by immunodiffusion assay is normal or increased, then the variant form of angioedema should be suspected. In this case, a functional C1-INH assay should be done to confirm the diagnosis although this test is not readily available (See Fig. 2).

There are three aspects to treatment in the management of HAE: 1) long term prevention; 2) short term prevention; and 3) treatment of acute episodes.

Long term prophylactic therapy is indicated in patients with frequent or debilitating episodes. Children usually do not require long term prophylaxis and preventive therapy is avoided during pregnancy. Antifibrinolytic agents and androgens have been used successfully in preventing attacks of angioedema although their mechanisms of action are not well understood. The antifibrinolytic agents which have received the most use are epsilonaminocaproic acid (EACA), (Amicar®), and tranexamic acid. These agents have been shown to reduce the frequency of attacks by 80%. Although it has not been proven, it is thought that antifibrinolytic agents act by inhibition of plasmin since plasmin has been implicated as a mediator in the evolution of an attack of angioedema via activation of C1. (Although this activation of C1 is presumably inhibited by antifibrinolytics, the levels of C4 and C2 remain low). The usual dose of EACA is 8-12 gm/day in 4 divided doses. The most common side effects are muscle pain, weakness, and postural hypotension. Myositis and rhabdomyolysis with elevated CPK and aldolase level may occur. The muscle enzymes return to normal when the dosage is decreased. Patients prone to thrombotic events are at risk with antifibrinolytic therapy.

Tranexamic acid is an EACA derivative and is a more potent antifibrinolytic agent. Unfortunately, it has been found to cause hepatitis in some patients and liver tumors and retinal changes in laboratory animals.

Androgen therapy has been in use for the last 20 years for long term prophylaxis of attacks. Methyltestosterone was one of the first agents used. Although quite effective, its masculinizing effects prohibit long term use in women. In addition, it has been noted to cause cholestatic jaundice and adverse effects on sperm production.

In order to reduce the masculinizing effects of

chronic hormonal therapy, danazol, which is an attenuated androgen has been used. Androgens have been found to raise the serum levels of C1-INH and C4 most likely by inducing synthesis of a normal C1-INH protein. Danazol has been shown to cause a threefold to fivefold increase of C1-INH in the serum. Although the long term effects of danazol are not known, it appears to have minimal side effects. It is tolerated well by females although weight gain, acne, and mild hirsutism are reported. The minimal effective dose of danazol is quite variable and ranges from 50-600 mg daily. The drug can be titrated according to serum measurements of C1-INH and C4. Patients on long term therapy should be monitored by physical examination, complete blood counts, and liver function tests every 6-12 months.

The second aspect of management of HAE involves short term preventative therapy. Patients requiring this form of treatment are those undergoing dental or surgical procedures. Although protocols vary, most employ the use of danazol and an antifibrinolytic agent for about one week prior to the procedure. Other studies suggest that fresh frozen plasma may be effective when given within 24 hours of the procedure. Presumably, fresh frozen plasma provides a source of C1-INH. It, however, also provides a source of C4 and C2 which are the substrates of activated C1. In addition, fresh frozen plasma carries the risk of transfusion hepatitis.

Since long term prophylactic therapy is not recommended in children, in patients with infrequent attacks, or in pregnancy, an alternative form of management is needed for acute episodes of angioedema. Antihistamines, sympathomimetics and steroids have not shown to be effective in the acute situation. Fresh frozen plasma has been used and appears to be effective although there is considerable controversy regarding its use.

Attention now has been turned to the infusion of a purified concentrate of C1-INH during acute attacks. To date the results of this treatment have been very good. C1-INH concentrate may be given in a single, slow infusion. It has been reported to cause higher levels of C1-INH in serum than levels produced by fresh frozen plasma. Patients also demonstrate an increased C4 activity. In addition, because of its method of preparation it has a lower risk of transfusion hepatitis. Its shelf life exceeds one year at 4°C. The current data indicate that the infusion of C1-INH concentrate may now be the treatment of choice for an attack of HAE in progress.

FINAL DIAGNOSIS HEREDITARY ANGIOEDEMA

*DR. ROBERT SUURMEYER: This case was diagnosed at the unusually early age of three. As you point out, often there is considerable delay in making the proper diagnosis. Would a determination of C4 be an appropriate screen for children presenting with croup syndrome to single out the cases of HAE?

DR. OLSEN: I feel the disease (HAE) is very rare and C4 determination in the common croup syndrome would not be cost effective.

**DR. LOUIS OFSTEIN: Are there other conditions in which the C4 is reduced?

***DR. FRANK FOSS: Any condition in which the classical pathway of complement is activated will cause a decrease in C4. This includes many disorders including common autoimmune disorders such as rheumatoid arthritis, lupus erythematosus, and certain renal diseases.

****DR. P. R. ASPAAS: The C2 kinin pathway is often implicated in the pathogenesis of HAE but there is no explanation as to why only localized edema occurs. According to recent articles, the long term use of danazol is promising with relatively minor side effects.

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See "WARNINGS"

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S D Council Meeting Highlights

The Council of the South Dakota State Medical Association met on Friday and Saturday, April 10 and 11, 1981, at the Ramada Inn, Sioux Falls, South Dakota. The following are major items of business transacted at this meeting.

1. REQUEST FROM SOUTH DAKOTA CHAPTER, AMERICAN ACADEMY OF PEDIATRICS FOR A RESOLUTION SUPPORTING ACTIONS WHICH WOULD REDUCE UNNECESSARY DEATHS ON SOUTH DAKOTA HIGHWAYS.

The Council tabled this request and directed the executive office to correspond with the Society indicating that the SDSMA agrees with the principles and concepts but has strong reservations on mandating such into law.

2. JOINT MEDICAL-LEGAL SEMINAR PROPOSED BY THE BAR ASSOCIATION. The Council recommended that a joint medical-legal meeting not be held at this time.

3. COMMENDATION. The Council commended Dr. Charles Hollerman, Dean of the USDSM, for his dedicated service on behalf of the Medical School and the success which the Medical School has demonstrated in graduating well trained clinicians.

4. INCREASE IN COUNCIL SIZE. It was noted that both the Aberdeen and Sioux Falls Districts are eligible to seat one additional Councilor for 1981-82.

5. APPOINTMENT TO THE BOARD OF DIRECTORS OF THE SOUTH DAKOTA MEDICAL SCHOOL ENDOWMENT ASSOCIATION. The Council appointed the following to serve a one year term on the Endowment Board: T. H. Sattler, M.D.; Warren Jones, M.D.; Robert Giebink, M.D.; Joseph Hamm, M.D.; Bruce Allen, M.D.; G. E. Tracy, M.D.; and Bruce Lushbough, M.D.

6. APPOINTMENT TO SODAPAC BOARD OF DIRECTORS. The Council appointed the following to serve a two year term on the SoDaPAC Board of Directors: W. R. Taylor, M.D.; Harvey Hart, M.D.; Mrs. Marie Hovland; T. J. Wrage, Jr., M.D.; Mrs. Virginia Stoltz; Curtis Wait, M.D.; Mrs. Barbara Wait; Mrs. Sandy Swanson; John Davis, M.D.; Robert Hohm, M.D.; Michael Haley, M.D.; Mrs. Marilyn Mabey; Durward Lang, M.D.; Courtney Anderson, M.D.; R. I. Porter, M.D.; Mrs. Marlys Porter; Wm. Quick, M.D.; Nathaniel Whitney,

M.D.; A. J. Barrett, M.D.; Mrs. Mary Ann Harris; R. G. Nemer, M.D.; Lawrence Nelson, M.D.; and Mr. Les Kinstad.

7. **COMMEMORATION OF DR. FRED LEIGH.** The Council commemorated Dr. Fred Leigh for his past service and dedication to the State Medical Association.

8. **HEALTH PLANNING FOR SOUTH DAKOTA.** The Council approved the concept of flexible health planning for South Dakota at the local level and requested that the Long Range Planning Committee draft a model for health planning and public health financing and present this proposal to other concerned persons and to the Council at a later date.

9. **HONORARY MEMBERSHIP.** The Council voted honorary membership status to the following: F. U. Sebring, M.D.; M. P. Merryman, M.D.; A. A. Lampert, M.D.; Donald Breit, M.D.; V. R. Vonburg, M.D.; and R. E. Lemley, M.D.

10. **NORTH DAKOTA BLUE SHIELD SECOND OPINION PROGRAM.** The Council reaffirmed its previous policy that such a federal second opinion program is not necessary in South Dakota.

11. **MEDICARE "B" NEWSLETTER.** The Council referred this newsletter concerning the limitation of liability provision of the Medicare program and the plans of North Dakota Blue Shield for implementation of this provision to the SDSMA's legal counsel to determine the physicians' legal responsibilities with regard to this program.

12. **NOMINATIONS FOR A PHYSICIAN TO SERVE ON THE HSA BOARD OF DIRECTORS.** The Council submitted the following three nominees for consideration of appointment to the HSA Board: Kennon Broadhurst, M.D.; W. R. Taylor, M.D.; and Eldon Bell, M.D.

13. **NOMINATION FOR TERM ON THE SOUTH DAKOTA STATE BOARD OF MEDICAL AND OSTEOPATHIC EXAMINERS.** The Council recommended that Dr. R. C. Jahraus be reappointed to a five year term on the Board.

14. **CRIPPLED CHILDREN'S AND FAMILY PLANNING PROGRAMS.** Information was received that new contracts will be sent to physicians for these programs along with a letter explaining that physicians can accept payment from these programs and still bill the patient for the difference or, in the case of indigent patients, bill the county. ■

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SD

President's Page



A Century Of Service

A century of service to people. This could well be a time for reflection about those you consider your "heroes" of South Dakota medicine; those who taught you the art of medicine, the science of medicine, and those who spent time in organized medicine for the benefit of all.

I want to reflect on my medical heroes hoping you will do the same. John H. Calvert, M.D., Pierre, Nebraska, my uncle and in his 51st year in solo practice, has been the doctor in the family. Bill Jones, M.D., of Sturgis, was my family doctor while I was in high school and along with Joe Hamm, M.D., was a close family friend as I grew up. The unselfish caring for people of these three men became my inspiration to become a doctor.

My USD sophomore preceptorship was with G.J. Bloemendaal, M.D. in Ipswich and this proved to be an opportunity to spend a month with a master physician who was loved by his patients and cared for them in the same way. When he was honored by SDSMA for 50 years of service to medicine, I was proud to say I knew him and had learned from him. That time spent with him is as vivid as if it were

yesterday. As doctors, we need to know the art of medicine, keep up with the science of medicine, share the same with students, and command the respect of patient and colleague alike. "Doc" Bloemendaal has done that.

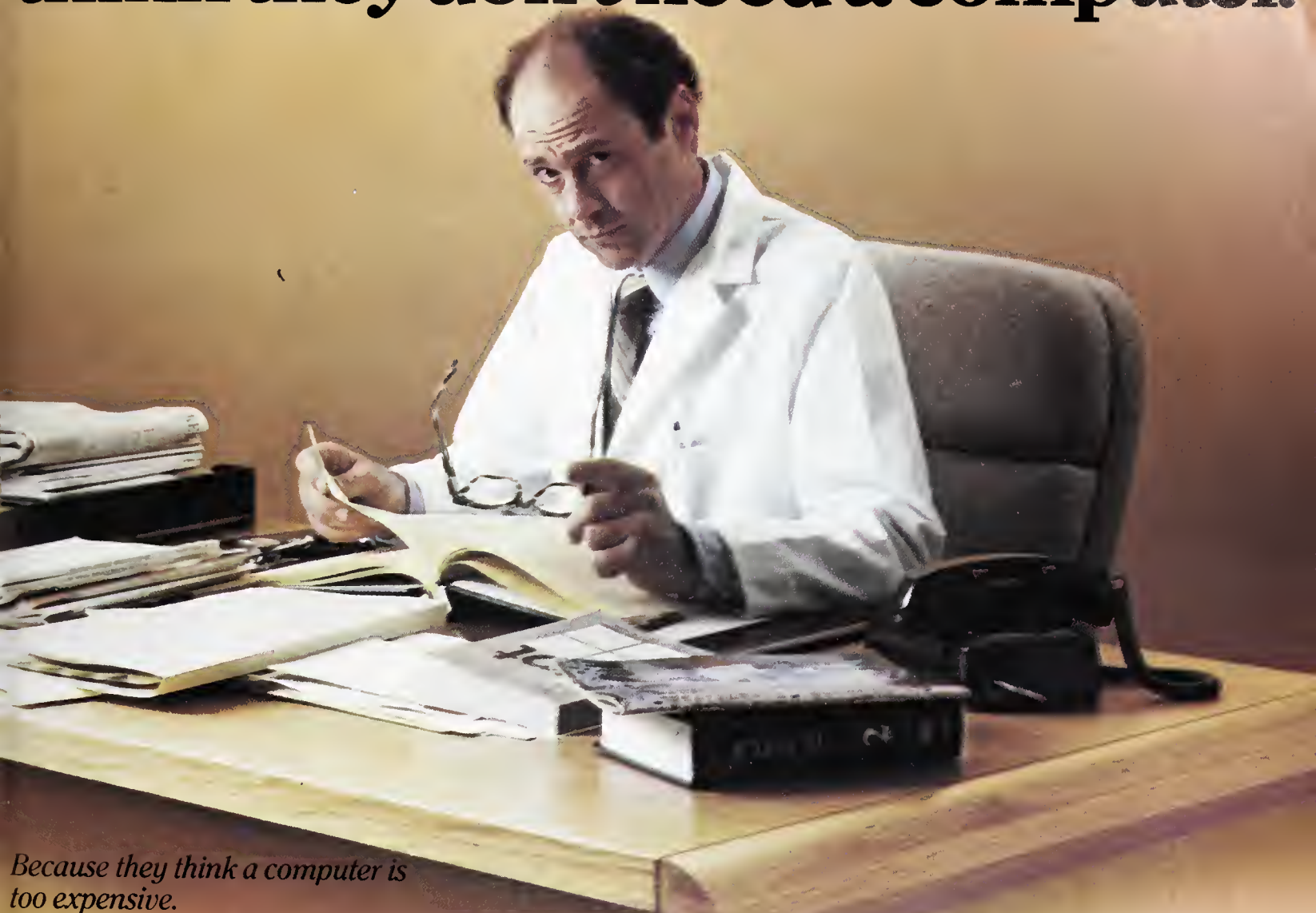
I know many others I consider heroes—all active in South Dakota medicine. Their goal has been, without exception, to give the people of South Dakota quality medical care.

I will strive for that goal as your president this year. As we work together, may we reflect on our "heroes" and rededicate ourselves to a second century of service to the people of South Dakota.

Sincerely yours,

Bruce Lushbough, M.D., President
South Dakota State Medical Association

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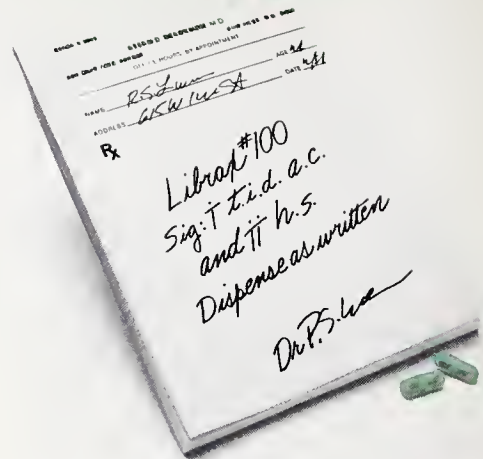
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As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression, suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants, causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated, avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment, blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, i.e., dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.



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Photograph of simulated gastric hypersecretion.

Although weight loss achieved in a weight control program varies from patient to patient, this simulated sequence of a professional model illustrates dramatically the benefits of a successful weight loss program.



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the #1 prescribed anorectic

An effective short-term adjunct in an indicated weight loss program

Overweight patients in certain diagnostic categories often require strict obesity control. Diethylpropion hydrochloride has been reported useful in obese patients with certain complications. While it is not suggested that Tenuate in any way reduces these complications in the overweight, it may have a useful place as a short-term adjunct in a prescribed dietary regimen. Tenuate should not be administered to patients with severe hypertension; see additional Warnings and Precautions on this page.

In uncomplicated obesity

Many patients, on the other hand, present with excess fat but no disease. While this condition is often termed uncomplicated obesity, complications of both a social and a psychologic nature may be distressingly real for the patients. In these cases, a short-term regimen of Tenuate can help reinforce your dietary counsel during the important early weeks of an indicated weight loss program.

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The anorectic effectiveness of diethylpropion hydrochloride is well documented. No less than 18 separate double-blind, placebo-controlled studies attest to its usefulness in daily practice.¹ And the unique chemistry of Tenuate provides "...anorectic potency with minimal overt central nervous system or cardiovascular stimulation."² Compared with the amphetamines, diethylpropion has minimal potential for abuse.

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(diethylpropion hydrochloride NF)

Tenuate Dospan® c
(diethylpropion hydrochloride NF)
controlled-release

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Brief Summary

INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.

CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect, rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. *Drug Dependence:* Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy:* Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children:* Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdose. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular:* Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System:* Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache, rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal:* Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic:* Urticaria, rash, ecchymosis, erythema. *Endocrine:* Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System:* Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous:* A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in mid-evening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in mid-morning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of January, 1980

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References: 1. Citations available on request from Merrell Dow Pharmaceuticals Inc., Cincinnati, Ohio 45215. 2. Hoekenga, M. T. et al: A comprehensive review of diethylpropion hydrochloride. In *Central Mechanisms of Anorectic Drugs*, S. Garattini and R. Samanin, Ed., New York. Raven Press, 1978, pp. 391-404.



SOUTH DAKOTA ACADEMY OF FAMILY PHYSICIANS
3001 South Holly Avenue
Sioux Falls, SD 57105

THE AMERICAN ACADEMY OF FAMILY PHYSICIANS
1740 West 92nd Street
Kansas City, Missouri 64114

The American Academy of Family Physicians is the national association of family doctors. It is the second largest national medical organization, with over 48,000 physician members in 50 states, D.C., Puerto Rico, and the Virgin Islands. Until October 3, 1971, it was known as the American Academy of General Practice. The name was changed in order to reflect more accurately the changing nature of primary health care.

The Academy was founded in 1947 to promote and maintain high standards for family doctors providing continuing comprehensive health care to the public. Other major purposes of the Academy include acknowledging and assuming responsible public advocacy in all health-related matters; preserving the right of free choice of physician to the patient; encouraging young people in preparing, qualifying, and establishing themselves in family practice, and assisting in providing postgraduate study courses for family physicians.

Realizing that the family doctor's effectiveness depends on sound, up-to-date continuing education, the founders wrote into the Bylaws the requirement that members must complete a minimum of 150 hours of approved continuing education every three years to retain membership.

This guarantee of competence is met through continuing education programs, publication or presentation of original scientific papers, medical school or postgraduate teaching, hospital residency training, etc. Accurate and current records are kept to insure that individual requirements are met; if they are not, the member is dropped from the rolls. The requirement, unique at time of origin, has through the years become a standard for an increasing number of other medical groups.

The Academy is governed by a Congress of Delegates composed of two delegates from each of the 54 constituent chapters as well as from resident and student groups. The Congress meets annually immediately prior to the Academy's Annual Scientific Assembly and has sole power to establish policies and define principles. These policies and programs are carried out between annual meetings by the Board of Directors and a number of standing and special commissions and committees. Delegates to the Congress elect the Board, which in turn appoints commission and committee members. Constituent chapters are similarly organized.

The Annual Scientific Assembly is the Academy's largest meeting for continuing education, drawing as many as 10,000 physicians and visitors.

The Academy was instrumental in the establishment of family practice, a derivative of classical general practice, as medicine's twentieth primary specialty. The AMA's Council on Medical Education and the independent American Board of Medical Specialties granted approval to a certifying board in family practice,

the basic structural requisite of a medical specialty, on February 8, 1969. Examinations have been given annually since that year and there are now about 22,000 diplomates of the American Board of Family Practice. About 73 percent of these are Academy members.

The Academy maintains national headquarters in Kansas City, Mo. with a professional staff of 125 persons. It publishes a monthly scientific magazine entitled **American Family Physician**, with a primary care physician circulation of 125,000, and a monthly all-member news and features publication entitled **AAFP Reporter**.

Delegates To Be Elected

As per the recommended new Bylaws, SDAFP will now elect delegates and alternate delegates for two year terms, to be staggered after the initial election (this year) so that one delegate and one alternate delegate will be elected each year for a two year term.

Nominated by the Board of Directors for election this year are:

Two Year Terms:	Delegate: L. H. Amundson, Sioux Falls
	Alternate Delegate: Herb Saloum, Tyndall
One Year Term:	Delegate: R. W. Friess, Sioux Falls
	Alternate Delegate: Ray Nemer, Gregory

This election will be held during the annual business meeting, August 14, 1981, in Rapid City.

These terms include service during the calendar year(s) following election, whereas elected chapter officers serve from one annual meeting to the next.

New Diplomates

The following active members of SDAFP are new diplomates, ABFP, having passed the two day certifying exam in 1980. Congratulations to:

Forrest S. Brady, Spearfish
Thomas J. Grau, Sioux Falls
Joel B. Huber, Redfield
Steven A. Massapust, Lead
Thomas J. Huber, Pierre
Richard R. McClafflin, Watertown
Lawrence Marc Model, Elk Point
Roger Dean Olsen, Sioux Falls
Robert Seidel, Sioux Falls
Larry B. VanderWoude, Sioux Falls

Plan Now To Attend

The AAFP's Annual Scientific Assembly, September 21-24, 1981, with special events for spouses. This event will be held in Las Vegas, Nevada.

Immediate And Delayed Tc-99M Glucoheptonate Brain Images

W. A. Boade, M.D.*

ABSTRACT:

Ninety-nine sequential isotope brain procedures (99m Tc-glucoheptonate) consisting of flow study views, immediate post-injection static views, and two hour post-injection static views were performed to evaluate the need for immediate post-injection static images. Seventeen cases were abnormal. Of these, three cases showed abnormalities in only the immediate post-injection static images. These abnormalities were CVA (two cases) and ventriculitis (one case). Therefore, it is possible that the immediate post injection static views may be useful in imaging necrotic or inflammatory lesions better than the two hour delayed images or the flow study images.

malities in only the immediate post-injection static images. These abnormalities were CVA (two cases) and ventriculitis (one case). Therefore, it is possible that the immediate post injection static views may be useful in imaging necrotic or inflammatory lesions better than the two hour delayed images or the flow study images.

INTRODUCTION

In most hospital-based nuclear medicine departments there are set procedures for all imaging studies. The time between injection and imaging generally is established by recommendations from the Nuclear Medicine literature.

Isotope brain imaging procedures are well established. Various radio-pharmaceutical preparations can be used. Nearly all of these utilize Technetium 99m which is attached to either glucoheptonate or DTPA (diethyl triamine penta acetic acid). The times at which static images are taken after injection vary greatly depending not only on the radio-pharmaceutical used, but also on the nature of the potential brain pathology.^{1,2}

PURPOSE

The purpose of this study was to determine the usefulness of immediate post-flow static images in detecting lesions of the central nervous system.

MATERIALS AND METHODS

Of 99 sequential radionuclide brain studies all were performed with posterior flow studies, immediate post-flow static images and two hour post-flow static images. The immediate post-flow static images consist of posterior, and right and left lateral views. The two hour post-flow static images consisted of anterior, posterior, right and left lateral and vertex views. The isotope used was 99m glucoheptonate. A standard single crystal Anger gamma camera was used along with data processing of the flow study information with a computer.

* Dept. of Nuclear Medicine, Laboratory of Clinical Medicine and Sioux Valley Hospital, Sioux Falls, SD.

Table I

Case #	Flow Study	Post Flow Static Views	2 hr. Static Views	Diagnoses
1.	negative	positive	positive	neoplasm
2.	negative	negative	positive	effusion, post meningitic
3.	*negative	positive	equivocal	ventriculitis
4.	negative	positive	positive	neoplasm
5.	*negative	positive	equivocal	CVA
6.	negative	negative	positive	probable neoplasm
7.	equivocal	equivocal	positive	degenerative CNS disease
8.	positive	equivocal	positive	CVA
9.	positive	negative	negative	sagittal sinus thrombosis
10.	positive	negative	negative	early CVA
11.	equivocal	negative	negative	early CVA
12.	positive	positive	positive	CVA
13.	negative	positive	positive	metastatic neoplasm
14.	positive	negative	positive	CVA
15.	*negative	positive	negative	CVA
16.	negative	positive	positive	neoplasm
17.	positive	positive	positive	neoplasm

*Three cases under discussion.

RESULTS

Of the 99 procedures performed, there were 17 abnormal studies. The abnormal studies consisted of abnormal flow study images alone, abnormal flow and static images or abnormal static images. There were three studies among the 17 abnormalities in which only a flow study abnormality was demonstrable. The remaining 14 studies showed 8 cases exhibiting static view abnormalities alone with normal flow study images. There were three cases in which the immediate post-flow static images exhibited abnormalities either not seen or seen to a lesser extent on the two hour post-flow static images. One of these cases was a CVA of the left posterior cerebral artery distribution. The second of these was a ventriculitis of an infant due to meningitis. The third case was an early CVA. There were no neoplastic lesions identified with the immediate post-flow static images which were not also demonstrated in the two hour post-flow images (Table I).

CONCLUSIONS

It appears possible, therefore, that the immediate post-flow static views are likely to pick up abnor-

malities of necrosis or inflammation which may not be seen or seen less intensely on the two hour post-flow static images. However, the value of the immediate post-flow static images is quite limited in visualizing neoplastic processes.

COMMENT

This series of 99 cases is quite small and probably would be more statistically accurate with more cases and more diversity of diagnoses. We did not have the opportunity to evaluate subdural hematomas. We did, however, see a subdural effusion in an infant who had been treated for meningitis. This abnormality was seen on only the two hour post-flow static images. We saw no abnormalities suspicious for A-V malformations.

REFERENCE

1. Tanasescu, DE, Wolfstein, RS, Brachman, MB, Waxman, AD, Early and delayed Tc-99m glucoheptonate brain scintigraphy: "Are routine early images indicated?" *J. Nucl. Med.*, 20:287-290, 1979.
2. Razzak, MA, Mudarris, F, Christie, JH, "Sensitivity of radionuclide brain imaging and computerized transaxial tomography in detecting subdural hematoma", *Clin. Nucl. Med.*, Vol. 5, No. 4, pp. 154-158, April 1980.



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WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as a failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of non-susceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section). Complete literature available on request from Professional Services Dept. PML.



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Combined Modality Treatment For Glioblastoma Multiforme

Donald G. Nordstrom, M.D.*

Malignant tumors of the brain have been noted to occur at the rate of 4.5 cases per 100,000 people, and of this group, 43% are described as malignant gliomas.¹ Within this category are tumors described as glioblastoma multiforme, malignant astrocytoma, or anaplastic astrocytoma.

Typically, the course of therapy for patients who have been diagnosed as having a glioblastoma multiforme has been either surgical resection and radiation therapy or radiation therapy alone. The dose has usually been 6000 rads in a course of six weeks. A review of selected articles by Sheline² shows a grade III grade tumors can have an expected five year survival of approximately 20%, but patients with grade IV lesions live less than five years regardless of their form of therapy.

High doses of radiation therapy have been attempted as a form of control for these lesions.³ Patients were treated with a total of 5000 to 6000 rads to the whole brain followed by a boost to the primary site as high as 8000 rads. Even with this technique, this has produced no increase in the long term survival of patients, as there seems to be no local tumor control despite the high doses utilized. Radiation necrosis of the brain is a definite consideration, this usually occurs between one and three years after the course of radiation therapy.

As a result, attempts to enhance the patients survival by combinations of chemotherapy or radiation sensitizers has been investigated. One of the sensitizers under investigation is Metronidazole

which has been shown to sensitize cells which are hypoxic, and has been used in clinical studies since 1973. Urtasun and colleagues^{4,5} have shown superior survival in those patients receiving Metronidazole in addition to radiation therapy. They show a 4½ month delay between relapse and subsequent death which was statistically significant.

Dianhydroglactitol has also been used in conjunction with radiation therapy,⁶ with doses of 5000 rads delivered to the brain either with or without the Dianhydroglactitol. With addition of the Dianhydroglactitol, approximately double the duration of survival, from 35 weeks to 67 weeks for patients with grade III and grade IV supratentorial astrocytomas is noted.

A recent article from the Brain Tumor Study Group has shown that patients receiving Carmustine given in conjunction with chemotherapy had the best survival when compared with patients receiving radiation therapy alone or in combination with Semustine or Semustine alone.⁷ However, the difference was not noted to be significant at the .05 level.

The Northcentral Cancer Treatment Group, a community based cooperative study program in the midwest, is also involved with evaluations of combination of radiation therapy and chemotherapy, APY. The NCCTG is an association of ten institutions with the collaboration of the Mayo Clinic investigating various therapeutic approaches for carcinomas. The current study for patients with primary brain tumors has a total of 32 patients on the study at this time and is continually recruiting new patients. This particular study is evaluating the combination of

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BCNU and Dibromodulcitol given in conjunction with radiation therapy as a first approach of therapy after biopsy or resection has been performed for these patients. The doses of radiation therapy are 4600 rads to the whole brain, followed by a boost of 1400 rads directed to the primary site. In addition, at the beginning of each course of therapy, the patients are randomized to receive either BCNU or DBD.

A second part of this study involves the use of VM-26 and VP-16 which is used when patients are noted to show progression of disease. The chemotherapy agents, BCNU and Dibromodulcitol are well tolerated by patients, common side effects are depression of the WBC and RBC count and mild nausea and vomiting.

This treatment study is available to patients in the eastern Dakotas as all of the oncologists in Sioux Falls are currently involved in the Northcentral Cancer Treatment Group. Any one of the oncologists could provide further information regarding this treatment protocol evaluating these drugs in combination with radiation therapy. It is hoped that sufficient numbers of patients with the diagnosis of glioblastoma multiforme can be entered on this study to test the effectiveness of these combinations of drugs with radiation therapy in an attempt to provide enhanced survival for patients with this diagnosis.

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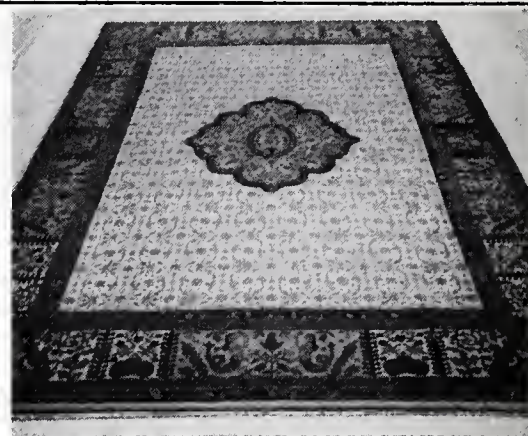
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EQUAGESIC—Abbreviated Summary

INDICATIONS: Based on a review of this drug by the National Academy of Sciences—National Research Council and on other information, FDA has classified the indications as follows:
"Possibly" effective for the treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease or tension headache.
Final classification of the less-than-effective indications requires further investigation.
The effectiveness of Equagesic in long-term use, i.e. more than four months, has not been assessed by systematic clinical studies. The physician should periodically reassess usefulness of the drug for the individual patient.

CONTRAINDICATIONS: Equagesic should not be given to individuals with a history of sensitivity or severe intolerance to aspirin, meprobamate, or ethoheptazine citrate.

WARNINGS: Careful supervision of dose and amounts prescribed for patients is advised, especially with those patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g., alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on or habituation to the drug. Where excessive dosage has continued for weeks or months, dosage should be reduced gradually rather than abruptly stopped, since withdrawal of a "crutch" may precipitate withdrawal reaction of greater proportions than that for which the drug was originally prescribed. Abrupt discontinuance of doses in excess of the recommended dose has resulted in some cases in the occurrence of epileptiform seizures.

Special care should be taken to warn patients taking meprobamate that tolerance to alcohol may be lowered with resultant slowing of reaction time and impairment of judgment and coordination.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with the use

of minor tranquilizers (meprobamate, chlorthalidopexide, and diazepam) during the first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physicians about the desirability of discontinuing the drug. Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breast-feeding patients, the drug's higher concentration in breast milk as compared to maternal plasma levels should be considered.

Preparations containing aspirin should be kept out of the reach of children. Equagesic is not recommended for patients 12 years of age and under.

PRECAUTIONS: Should drowsiness, ataxia, or visual disturbance occur, the dose should be reduced. If symptoms continue, patients should not operate a motor vehicle or any dangerous machinery. Suicidal attempts with meprobamate have resulted in coma, shock, vasomotor and respiratory collapse, and anuria. Very few suicidal attempts were fatal, although some patients ingested very large amounts of the drug (20 to 40 gm). These doses are much greater than recommended. The drug should be given cautiously, and in small amounts, to patients who have suicidal tendencies. In cases where excessive doses have been taken, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Hyperventilation has been reported occasionally. Any drug remaining in the stomach should be removed and symptomatic treatment given. Should respiration become very shallow and slow, CNS stimulants, e.g., caffeine, Metrazol, or amphet-

mine, may be cautiously administered. If severe hypotension develops, pressor amines should be used parenterally to restore blood pressure to normal levels.

ADVERSE REACTIONS: A small percentage of patients may experience nausea with or without vomiting and epigastric distress. Dizziness occurs rarely when meprobamate and ethoheptazine citrate with aspirin is administered in recommended dosage. The meprobamate may cause drowsiness but, as a rule, this disappears as therapy is continued. Should drowsiness persist and be associated with ataxia, this symptom can usually be controlled by decreasing the dose, but occasionally it may be desirable to administer central stimulants such as amphetamine or mephentermine sulfate concomitantly to control drowsiness.

A clearly related side effect to the administration of meprobamate is the rare occurrence of allergic or idiosyncratic reactions. This response develops, as a rule, in patients who have had only 1-4 doses of meprobamate and have not had a previous contact with the drug. Previous history of allergy may or may not be related to the incidence of reactions.

Mild reactions are characterized by an itchy urticarial or erythematous, maculopapular rash which may be generalized or confined to the groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema, and fever have also been reported.

More severe cases, observed only very rarely, may also have other allergic responses, including fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case), and hyperthermia. Treatment should be symptomatic such as administration of epinephrine, antihistamine, and possibly hydrocortisone. Meprobamate should be stopped, and re-institution of therapy should not be attempted.

Rare cases have been reported where patients receiving meprobamate suffered from aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia. In nearly every instance reported, other toxic agents known to have caused these conditions have been associated with meprobamate. A few cases of leukopenia during

continuous administration of meprobamate are reported, most of these returned to normal without discontinuation of the drug.

Impairment of accommodation and visual acuity has been reported rarely.

OVERDOSE: Two instances of accidental or intentional significant overdose with ethoheptazine citrate combined with aspirin have been reported. These were accompanied by symptoms of CNS depression, including drowsiness and light-headedness, with uneventful recovery. However, on the basis of pharmacological data, it may be anticipated that CNS stimulation could occur. Other anticipated symptoms would include nausea and vomiting. Appropriate therapy of signs and symptoms as they appear is the only recommendation possible at this time. Overdosage with ethoheptazine combined with aspirin would probably produce the usual symptoms and signs of salicylate intoxication. Observation and treatment should include induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions.

DESCRIPTION: Each Equagesic tablet contains 150 mg meprobamate, 75 mg ethoheptazine citrate and 250 mg aspirin.

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*This drug has been evaluated as possibly effective for this indication.

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WYGESIC—Abbreviated Summary

INDICATION: For the relief of mild-to-moderate pain.

CONTRAINDICATION: Hypersensitivity to propoxyphene or to acetaminophen.

WARNINGS: CNS ADDITIVE EFFECTS AND OVERDOSAGE: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, or other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. Toxic effects and fatalities have occurred following overdoses of propoxyphene alone or in combination with other CNS depressants. Most of these patients had histories of emotional disturbances or suicidal ideation or attempts, as well as misuse of tranquilizers, alcohol, or other CNS-active drugs. Caution should be exercised in prescribing large amounts of propoxyphene for such patients (see **Management of Overdosage**).

DRUG DEPENDENCE: Propoxyphene can produce drug dependence characterized by psychic dependence and less frequently physical dependence and tolerance. It will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics. The abuse liability of propoxyphene is qualitatively similar to codeine's although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine.

USAGE IN AMBULATORY PATIENTS: Propoxyphene may impair the mental and/or physical abilities required for potentially hazardous tasks (e.g., driving a car or operating machinery). Patients should be cautioned accordingly.

USAGE IN PREGNANCY: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. **INSTANCES OF WITHDRAWAL SYMPTOMS IN THE NEONATE HAVE BEEN REPORTED FOLLOWING USAGE DURING PREGNANCY.** Therefore, propoxyphene should not be used in pregnant women unless, in the

judgement of the physician, the potential benefits outweigh the possible hazards.

USAGE IN CHILDREN: Propoxyphene is not recommended for children because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric group.

PRECAUTIONS: Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine. The CNS depressant effect of propoxyphene may be additive with other CNS depressants, including alcohol.

ADVERSE REACTIONS: The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting. These seem more prominent in ambulatory than in nonambulatory patients, some of these reactions may be alleviated if the patient lies down. Other adverse reactions include constipation, abdominal pain, skin rashes, light-headedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances. The chronic ingestion of propoxyphene in doses over 800 mg per day has caused toxic psychoses and convulsions. Cases of liver dysfunction have been reported.

DRUG INTERACTIONS: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, and other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended (see **Warnings**). Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine.

MANAGEMENT OF OVERDOSAGE: **SYMPTOMS:** The manifestations of serious overdosage with propoxyphene are similar to those of narcotic overdosage and include respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, cyanosis), extreme somnolence progressing to stupor or coma, pupillary constriction, and circulatory collapse. In addition to these characteristics, which are reversed by narcotic antago-

nists such as naloxone, there may be other effects. Overdoses of propoxyphene can cause delay of cardiac conduction as well as focal or generalized convulsions, a prominent feature in most cases of severe poisoning. Cardiac arrhythmias and pulmonary edema have occasionally been reported, and apnea, cardiac arrest, and death have occurred.

Symptoms of massive overdosage with acetaminophen may include nausea, vomiting, anorexia, and abdominal pain, beginning shortly after ingestion and lasting for 12 to 24 hours. However, early recognition may be difficult since early symptoms may be mild and nonspecific. Evidence of liver damage is usually delayed. After the initial symptoms, the patient may feel less ill, however, laboratory determinations are likely to show a rapid rise in liver enzymes and bilirubin. In case of serious hepatotoxicity, jaundice, coagulation defects, hypoglycemia, encephalopathy, coma and death may follow. Renal failure due to tubular necrosis, and myocardialopathy, have also been reported.

Ingestion of 10 grams or more of acetaminophen may produce hepatotoxicity. A 13-gram dose has reportedly been fatal.

TREATMENT: Primary attention should be given to the reestablishment of adequate respiratory exchange through provision of a patent airway and institution of assisted or controlled ventilation. The narcotic antagonists, naloxone, nalorphine, and levallorphan, are specific antidotes against the respiratory depression produced by propoxyphene. An appropriate dose of one of these antagonists should be administered preferably I.V., simultaneously with efforts at respiratory resuscitation and the antagonist should be repeated as necessary until the patient's condition remains satisfactory. In addition to a narcotic antagonist the patient may require careful titration with an anticonvulsant to control seizures. Analeptic drugs (e.g., caffeine or amphetamine) should not be used because of their tendency to precipitate convulsions.

Oxygen, IV fluids, vasopressors and other supportive measures should be used as indicated. Gastric lavage may be helpful. Activated charcoal can absorb a significant amount of ingested propoxyphene. Dialysis is of little value in poisoning by propoxyphene alone. Acetaminophen is rapidly absorbed and efforts to remove the drug from the body should not be delayed. Copious gastric lavage and/or induction of emesis may be indicated. Activated charcoal is probably ineffective unless administered almost immediately after acetaminophen ingestion. Neither forced diuresis nor hemodialysis appears to be effective in removing acetaminophen. Since acetaminophen in overdose may have an antidiuretic effect and may produce renal damage, administration of fluids should be carefully monitored to avoid overload. It has been reported that mercaptamine (cysteine) or other thiol compounds may protect against liver damage if given soon after overdosage (8-10 hours). N-acetylcysteine is under investigation as a less toxic alternative to mercaptamine, which may cause anorexia, nausea, vomiting, and drowsiness. Appropriate literature should be consulted for further information (JAMA 237:2406-2407, 1977). Clinical and laboratory evidence of hepatotoxicity may be delayed up to one week. Acetaminophen plasma levels and half-life may be useful in assessing the likelihood of hepatotoxicity. Serial hepatic enzyme determinations are also recommended.

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Family Physician Needs For South Dakota—1990

L. H. Amundson, M.D.*

Preface

Family Physician

The family physician provides health care in the discipline of family practice. His training and experience qualify him to practice in the several fields of medicine and surgery.

The family physician is educated and trained to develop and bring to bear in practice unique attitudes and skills which qualify him or her to provide continuing, comprehensive health maintenance and medical care to the entire family regardless of sex, age or type of problem, be it biological, behavioral or social. This physician serves as the patient's or family's advocate in all health-related matters, including the appropriate use of consultants and community resources.

Official Definition

American Academy of Family Physicians

As the South Dakota State Medical Association completes 100 successful years of helping to promote quality medical care in the state, it seems appropriate to consider physician needs in South Dakota as we enter the second century of service. The specific intent of this paper is to define the need for family physicians. Family practice is being done every day by more than 60,000 physicians across the United States¹. Of the 793 physicians across the living in South Dakota, 221 are doing family practice (Tables I, II). It is this latter number upon which we will focus.

*Secretary-Treasurer, South Dakota Academy of Family Physicians; Associate Director, Sioux Falls Family Practice Residency Program; Professor of Family Medicine, University of South Dakota School of Medicine.

Table I
PHYSICIANS LICENSED
AND LIVING IN
SOUTH DAKOTA
DECEMBER 1980

Total M.D. and D.O.	793*
Retired	32
Residents in Training	26
Federal Employees	45
Full Time Teaching	12
Non-Patient Care	67
Hospital Based	
Total Patient Care Physicians (Non-Federal)	611
*24 Females (3.8%)	(496 in 1974)

Table II
NON-FEDERAL
PATIENT CARE PHYSICIANS
IN
SOUTH DAKOTA
DECEMBER 1980

Family Physicians	144
General Practitioners	67
Osteopathic Practitioners	10
	221 - 36%
General Internal Medicine	48
General Pediatrics	27
	75 - 12%
Other Patient Care Physicians	315 - 52%
TOTAL	611 - 100%

Table III
SOUTH DAKOTA POPULATION AND PHYSICIAN DATA

Year	South Dakota Population	South Dakota		National		Ranking by State
		Total M.D.s	Non-Federal M.D.s	Total M.D.s	Non-Federal M.D.s	
1930	692,849		120/100,000 pop.*		134/100,000 pop.	
1940	642,961		142/100,000 pop.*		141/100,000 pop.	
1950	652,740					
1960	680,514					
1970	666,257 (-2%)					51/51
1980	690,178 (+3.6%)	793**	100/100,000 pop.	440,000**	173/100,000 pop.	51/51
1990	715,024 (est. +3.6%)			594,000***	242/100,000 pop.***	

*Predominately Patient Care General Practitioners, Non-Federal

**77% Patient Care Physicians

***HHS Predictions

Background

The 1974 South Dakota Legislature formally created "at the University of South Dakota a family practice oriented four-year M.D. degree granting School of Medicine which shall be under the control of the Board of Regents." A two-year Basic Science Medical School had been in existence since 1907². The change was in response to South Dakota having the poorest physician/population ratio in the country (Table III), an aging physician population (average age 55), and a geographic maldistribution of physicians. Rural South Dakota clearly needed more family physicians.

The AMA reports public satisfaction with medical care quality remains high, 88% saying they were "very or fairly satisfied" with the care they received in 1978. Only 33% of the public would approve of being treated by a group of physicians instead of a personal physician according to latest surveys commissioned by the American Medical Association and conducted by the Gallup organization³. During the last decade average practice expenses for physicians have increased faster than average gross income, and average real net income for physicians fell slightly. The average waiting time to obtain an appointment and waiting time in the office declined^{3,4}.

General and family physicians and osteopathic general physicians will increase from about 60,000 to 90,000 by the end of the 1980s. The 24,000 general pediatricians in practice today will grow to 38,000 by 1990, and the specialty of internal medicine will experience a 51% increase in the number of practitioners during this decade⁵. This Rand study of 1977 showed that the number of specialists tripled between 1960 and 1970 and revealed that in 1977 there were five certified internal medicine spe-

cialists per 100,000 people in cities of 20,000 to 30,000 in the United States. The data further showed that, whereas in 1960 fewer than one-third of communities with 20,000 to 30,000 residents had Board certified specialists such as internists, pediatricians, obstetrician-gynecologists, radiologists, and surgeons, 17 years later "a full complement of the five" specialists was found in more than 70% of the communities.

Congress has supported family medicine for ten years on the assumption that such support would alleviate maldistribution and improve access to care⁶. The Graduate Medical Education National Advisory Committee (GMENAC) report, released in late 1980, states that family practice residency programs should be supported since they "tend to train providers who are more likely to choose to practice in underserved areas." A more "vigorous and imaginative emphasis" should be placed on ambulatory care training experiences. Family practice programs, at least for the near term, "should be given special attention" because of difficulty in financing them from ambulatory care revenues²¹.

Portland, Oregon's physician to population ratio is now 1 to 167³.

The Setting

The need for more rural physicians has long been a problem faced by the South Dakota State Medical Association⁷. A lack of doctors in all but eight of the largest cities in the state was noted in 1945. This "lack and improper distribution of physicians" was considered the Association's "most serious problem" by its president F. S. Howe in 1947.

Currently, there are 27 general pediatricians in South Dakota. Forty-eight general internists practice in South Dakota, 7 per 100,000 population, which is similar to large metropolitan area ratios in

1960⁵. Twenty-four women (3.8%) practice in South Dakota (Table I), compared with 11% nationally³.

Department of Health, Education and Welfare (now Health and Human Services) figures show that the average M.D. visits per year per person is just over 5 office visits. Accordingly, 3,450,000 office visits are made per year in South Dakota⁸. A national study showed that 234,660,000 office visits were made to family physicians and general practitioners in 1975⁹. Family physicians in South Dakota are responsible for over 4,000 hospital admissions per month¹⁰. More than 400 patients are cared for in or referred to a tertiary care medical center per month¹¹ (Table IV). These same 221 family physicians make 2,334 consultations and referrals to their colleagues per month, including ambulatory and hospital patients¹³.

A typical family physician spends 49 hours weekly in direct patient care, 35 of those in the office, and sees approximately 132 patients a week, usually by appointment. Patient profiles are as follows: 15.1% under 15 years of age; 14.2% 15 to 24 years; 21.5% 25 to 44 years; 24.7% 45 to 64 years; and 24.4% 65 years and older. The mean patient waiting time is 21.7 minutes¹². Previous studies have shown that health care problems seen in South Dakota are similar to those in other parts of the country¹⁴. This study, done at the residency program, shows that family practice residents are being trained to care for common health problems. In addition, continuity of care, of value to the extent that it has an

Table IV
ECOLOGY OF MEDICAL CARE

Adult population at risk	1000
Adults reporting one or more illnesses or injuries per month	750
Adults consulting a physician one or more times per month	250
Adult patients admitted to a hospital per month	9
Adult patients referred to another physician per month	5
Adult patients referred to a university medical center per month	1

Reference:

White, K., Williams, T. F. and Greenberg, B.G.: The Ecology of Medical Care. New Engl J Med, 265:886-891, 1961.

impact on the outcome of care, the prevention or reduction of physical, mental, or social disabilities, the satisfaction of patients, and the cost of care is also taught. Studies of continuity of care generally have reinforced the belief that it is an important goal in medical care¹⁵. Continuity of care can be taught, and is an integral part of most family practice training programs¹⁶.

Forty-five family physicians have been trained in the Sioux Falls Family Practice Residency program and 22 are practicing in the state (Table V). Of those currently in training, more than one-third are native South Dakotans and nearly 25% of current family practice residents in training in South Dakota are women (Table VI).

Of the first four graduating classes of M.D.s from USDSM, 23.6% of graduates have entered family practice residencies, 44% of this group entering the Sioux Falls program¹⁷. USDSM has a 25% female

TABLE V
SFFPR PROGRAM
GRADUATES
7 YEAR STUDY
(1975-1981*)

Total Number	45
Native South Dakotans	16
Graduates of USDSM 2 Year School	12
Graduates of USDSM 4 Year School (2 classes possible)	7
Number of Females	2 (4.5%)
Practicing in South Dakota	22 (48%)
Practice - Bordering States	11 (24%)
Practice Elsewhere	12 (28%)

*Effective July 1, 1981

Table VI
SFFPR PROGRAM
CURRENT RESIDENTS
JANUARY 1981

Total Number	33
Year I	9
Year II	12
Year III	12
Native South Dakotans	13
Graduates of USDSM 2 Year School	2
Graduates of USDSM 4 Year School (Three Classes Possible)	10
Number of Females	8 (24%)

medical student population, comparable to 1979 figures which showed that 26.5% of all U.S. medical students were women.

Methodology

For purposes of this study, 1980 U.S. census figures were used¹⁸. Family physicians, general practitioners and osteopathic physicians are included in the classification of family physicians in this study. This study is based upon a ratio of one family physician to 2,500 population. This ratio has been

used as a goal in several studies throughout the United States²⁰. Another reason for choosing the 1 to 2,500 ratio as a goal is that this reflects the approximate ratio of family physicians to population in those industrialized nations having a comparable health care delivery system, but without acute medical manpower problems. In the past decade, the nationwide family physician to population ratio has been 1 to 3,300, although varying widely from state to state²⁰. In 1980 this ratio was 1 to 3,125 in South Dakota (Table VII). It is possible that the

Table VII
FAMILY PHYSICIAN NEEDS IN SOUTH DAKOTA-1990

County	SD Population/County in			Number of FPs/County in 1980	Ideal Number FPs			Number of FPs Needed by 1990		Probable Practice Sites
	1960	1970	1980		1980 1:2500	1990 1:2500	1:2000	1:2500	1:2000	
I. BLACK HILLS										
A. Western										
Butte	8,592	7,825	8,373	2				43 Now		Belle Fourche
Custer	4,906	4,698	5,868	1				60% = 26		Custer
Fall River	10,688	7,505	8,431	3						Deadwood
Harding	2,371	1,855	1,691	0						Edgemont
Lawrence	17,075	17,453	18,386	17						Hot Springs
Meade	12,044	16,618	20,684	5						Lead
Pennington	58,195	59,349	70,253	13						Lemmon
Perkins	5,977	4,769	4,570	2						Rapid City
			138,256	43	55	57	72	31	46	Spearfish
										Sturgis
		(Est. Pop. 1990)	143,233							
II. "WEST RIVER"										
A. West River Prairie										
Bennett	3,053	3,088	3,031	2				9 Now		Kadoka
Haakon	3,303	2,802	2,781	1				60% = 5		Martin
Jackson	1,985	1,531	3,189	1						Mission
Jones	2,066	1,882	1,445	0						Murdo
Mellette	2,664	2,420	2,214	0						Philip
Shannon	6,000	8,198	9,407	2						Pine Ridge
Todd	4,661	6,606	7,329	2						Rosebud
Tripp	8,761	8,171	7,162	1						Winner
Washabaugh	1,042	1,389	1,439	0						
Ziebach	2,495	2,221	2,288	0						
			40,294	9	16	17	20	12	15	
		(Est. Pop. 1990)	41,744							
III. "THE RIVER"										
A. Upper Missouri River										
Campbell	3,531	2,866	2,237	0				9 Now		Eagle Butte
Corson	5,798	4,994	5,125	1				60% = 5		Gettysburg
Dewey	5,257	5,170	5,350	1						Hoven
Potter	4,926	4,449	3,686	3						Mobridge
Walworth	8,097	7,842	7,006	4						
			23,404	9	9	10	12	5	7	
		(Est. Pop. 1990)	24,246							
B. Central Missouri River										
Hughes	12,725	11,632	14,211	9				9 Now		Pierre
Hyde	2,602	2,515	2,084	0				60% = 5		
Stanley	4,085	2,457	2,542	0						
Sully	2,607	2,362	1,991	0						
			20,828	9	9	9	11	4	6	
		(Est. Pop. 1990)	21,626							

C. Lower Missouri River											
Brule	6,319	5,870	5,169	3	13 Now					Armour Chamberlain Gregory Murdo Platte Presho Wagner	
Buffalo	1,547	1,739	1,795	0	60% = 8						
Charles Mix	11,785	9,994	9,693	3							
Douglas	5,113	4,569	4,179	2							
Gregory	7,399	5,710	5,979	5							
Lyman	4,428	4,060	3,853	8							
			30,668	13	13	13	16	5	8		
	(Est. Pop. 1990)		31,772								
IV. NORTH EAST											
A. Aberdeen											
Brown	34,106	36,920	36,901	7	16 Now					Aberdeen Eureka Faulkton Redfield	
Edmunds	6,079	5,548	5,174	2	60% = 10						
Faulk	4,397	3,893	3,293	1							
McPherson	5,821	5,022	4,029	2							
Spink	11,706	10,595	9,207	4							
			58,609	16	23	24	30	14	20		
	(Est. Pop. 1990)		60,718								
B. North East Lake Region											
Clark	7,134	5,515	4,889	0	21 Now					Britton Milbank Sisseton Watertown Webster	
Codington	20,220	19,140	20,898	10	60% = 13						
Day	10,516	8,713	8,106	2							
Grant	9,913	9,005	8,999	2							
Hamlin	6,303	5,172	5,248	1							
Marshall	6,663	5,965	5,410	2							
Roberts	13,190	11,678	10,897	4							
			64,447	21	26	27	33	14	20		
	(Est. Pop. 1990)		66,767								
V. EAST CENTRAL											
A. Huron-Mitchell											
Aurora	4,749	4,183	3,614	0	20 Now					Huron Miller Mitchell Wessington Springs	
Beadle	21,682	20,877	19,210	8	60% = 12						
Davidson	16,681	17,319	17,811	7							
Hand	6,712	5,883	4,956	4							
Hanson	4,584	3,781	3,385	0							
Jerauld	4,048	3,310	2,921	3							
Sanborn	4,641	3,697	3,206	0							
			55,103	20	22	23	29	9	15		
	(Est. Pop. 1990)		57,086								
B. Brookings-Madison											
Brookings	20,046	22,158	24,263	6	15 Now					Arlington Brookings Clear Lake De Smet Flandreau Lake Preston Madison	
Deuel	6,782	5,686	5,271	2	60% = 9						
Kingsbury	9,227	7,657	6,684	3							
Lake	11,764	11,456	10,766	3							
Miner	5,398	4,454	3,741	0							
Moody	8,810	7,622	6,682	1							
			54,407	15	23	24	30	15	21		
	(Est. Pop. 1990)		59,473								
VI. SOUTH EAST											
A. Sioux Falls-Yankton											
Bon Homme	9,229	8,577	8,061	2	66 Now					Beresford Brandon Bridgewater Canton Dells Rapids Elk Point Freeman Lennox Parker Parkston Scotland Sioux Falls Tyndall Vermillion Viborg Yankton	
Clay	10,810	12,923	13,693	3	60% = 40						
Hutchinson	11,085	10,379	9,357	7							
Lincoln	12,371	11,761	14,111	3							
McCook	8,268	7,246	6,445	3							
Minnehaha	86,575	95,209	110,029	39							
Turner	11,159	9,872	9,260	3							
Union	10,197	9,643	10,745	1							
Yankton	17,551	19,039	18,933	5							
			200,634	66	80	83	104	43	64		
	(Est. Pop. 1990)		207,859								
			690,178	221	276	286	357	155	220		
	(Est. Pop. 1990)		715,024 (+ 3.6%)				Average = 19/yr 1980-1990				

United States will wish to reach a ratio even lower (1 to 2,000 or 1 to 1,800), as its final goal. No attempt is made in this study to delineate physician needs in specialties other than family practice.

It should be stressed that these ratios apply to the population as a whole and do not deal with distribution. It is conceivable that a given area could have this ratio of family physicians to population, but still have areas within it where care is difficult to obtain. To help delineate area needs for family physicians in South Dakota, the state has been arbitrarily broken down into sections for study purposes. These areas have been so designated, based upon geography and population density. Six basic areas have been designated, with further delineation in three of the areas, as noted in Table VII and Figure 1. In the future, a new basic unit called the "Functional Medical Service Area" (FMSA) may be used for medical manpower planning because a recent study finds geopolitical boundaries—such as states or counties—inadequate for analysis of medical services²¹. The FMSA is described as "a small geographic unit with-

in which the large majority of the residing population receives specified health services." Appreciating the potential for change, this study still looks at county and state population figures during the past decades and projects needs for the next ten years.

National figures show that no more than 60% of present physicians practicing will be available for practice in 1990, due to retirement, death, and other forms of attrition²⁰. Therefore, 60% of the 221 family physicians currently in practice in the state, 133, will still be in practice in 1990 (Table VII).

It is the above premises that lead to this study, i.e. an attempt to define the number of new family physicians to be trained and in practice, and where these family physicians will have to be added within the state of South Dakota in the next decade, to provide at least a 1 to 2,500 ratio by 1990.

Results

To have attained a 1 to 2,500 family physician/population ratio in 1980 we would have needed 276 family physicians in South Dakota, 55 more than

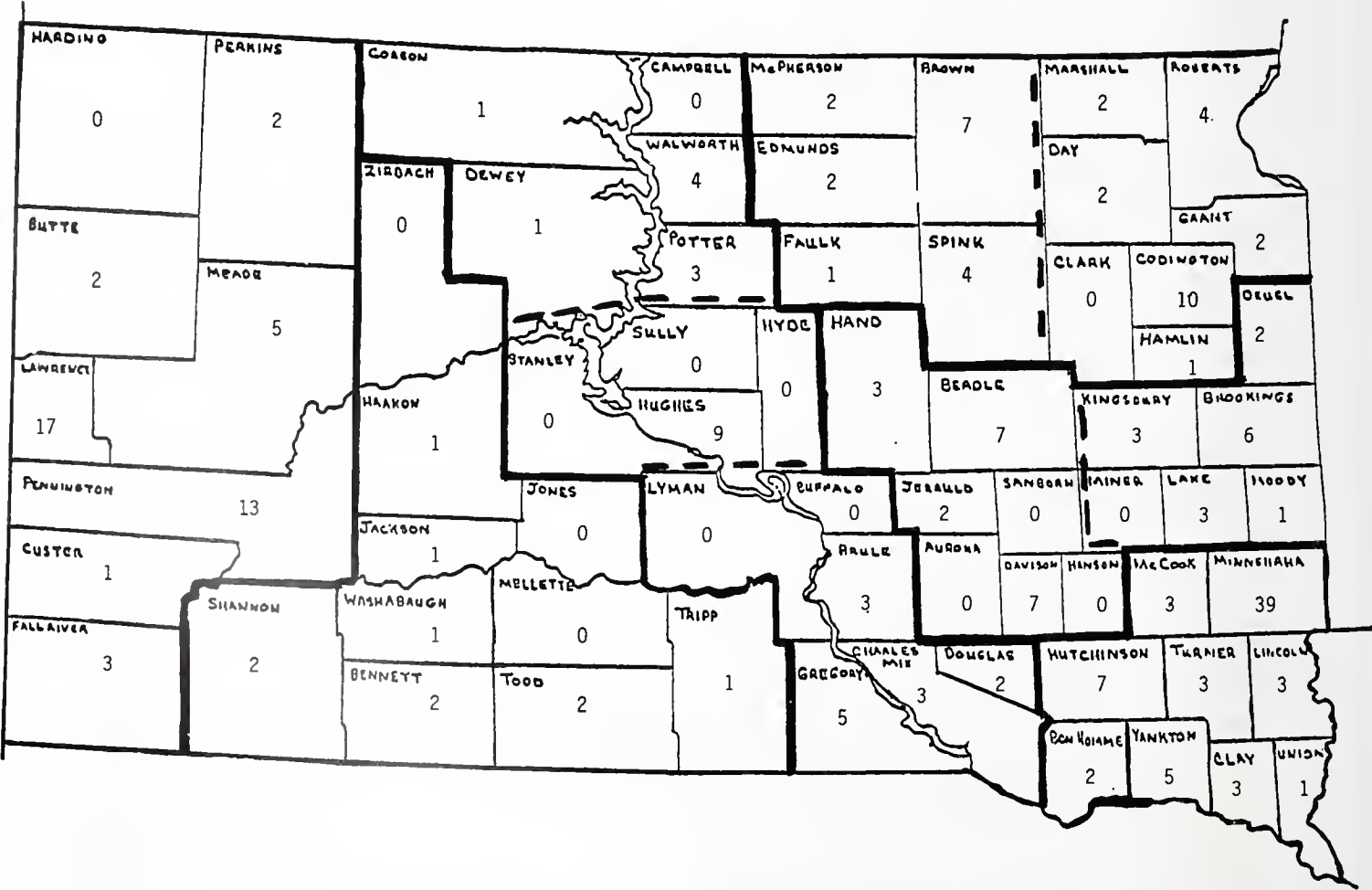


Figure 1
South Dakota
Number of Family Physicians/General Practitioners/Osteopathic Physicians, By County. December 1980

currently in practice. Using a projected 3.6% increase in population during the 1980s, similar to the 3.6% growth in the 1970s, the number of family physicians needed reaches 286 or more by 1990. It is anticipated that most of the population growth will occur in a few of the more "urbanized" areas of South Dakota. Thirty-seven percent of the physicians trained will be needed in areas I, II, III, Table VII.

According to this study, the number of family physicians to be trained will be 155 if we are to attain a 1 to 2,500 population ratio and 220 for a population ratio of 1 to 2,000. An average of these two figures shows a need for 19 per year during the coming decade if we are to reach 1990 predicted ratios (Table VII).

Discussion

There has been remarkable expansion of the medical education capacity of this country during the past two decades. Expansion continues, and with the declining rate of population growth, many knowledgeable people now project an excess aggregate physician supply²¹. Projections suggest that personal physicians who can serve as principal providers of care will probably be available for 85% of the American population in 1985 and 94% by 1990²³. GMENAC predicts surpluses by 1990 in general internal medicine and general pediatrics. An almost negligible surplus—both numerically and proportionately—is anticipated in family medicine. No further increase in the number of non-physician health care providers trained should occur. Research in and requirements for such providers needs to be done in view of the projected general physician surplus²¹.

A larger supply of physicians has some positive aspects: 1) Physicians will be able to spend more time with patients, a luxury not always possible in past years. 2) The art of medicine—the personal healing touch—sometimes lost in the rush to care for a great number of people—may be revived. 3) Some of the pressures of an overly heavy patient load will be eased. 4) Physicians may have more time for themselves and their families³.

The Rand Study shows that geographic maldistribution of physicians continues to be a major concern of public policy⁵. The bottom line in GMENAC's report states that the biggest problem to solve in 1990 will still be geographic maldistribution, realizing that specialty maldistribution needs attention now²¹. A recent administration report to Congress said the anticipated favorable balance between aggregate national supply and need in 1990 in most disciplines does not necessarily mean that geographic distribution problems will be resolved³.

"To solve geographic inequities will require some leeway in the supply to encourage potentially excess health personnel to locate in areas that would not otherwise get the manpower required," another report stated²³.

There are many underserved communities, especially in rural areas and inner cities; the geographic maldistribution and the perception of a public preference for more generalist physicians continue to fuel expansion of family practice training programs²². Family medicine will need to demonstrate that its graduates are contributing to the improvement of access to quality health care for all Americans; and that is thereby addressing our national health priorities and is deserving of further taxpayer support. From results to date, family medicine can be expected to demonstrate that the logic of governmental support has been correct and the faith has been well-founded⁶.

An attempt has been made in this study to delineate the need for family physicians in South Dakota in 1990, using geographic and population patterns to reach some conclusions. If at least 19 family physicians need be trained each year during the 1980s, it is incumbent upon South Dakota to be significantly involved in providing educational opportunities and financial support for this number. This number would equal at least 40% of the total graduating class from USDSM annually. These seem like appropriate "family practice oriented" goals for the University of South Dakota School of Medicine and its affiliated residency program(s).

Postscript

Family Practice

Family practice is comprehensive medical care with particular emphasis on the family unit, in which the physician's continuing responsibility for health care is limited neither by the patient's age or sex nor by a particular organ system or disease entity.

Family practice is the specialty in breadth which builds upon a core of knowledge derived from other disciplines—drawing most heavily on internal medicine, pediatrics, obstetrics and gynecology, surgery and psychiatry—and which establishes a cohesive unit, combining the behavioral sciences with the traditional biological and clinical sciences. The core of knowledge encompassed by the discipline of family practice prepares the family physician for a unique role in patient management, problem solving, counseling and as a personal physician who coordinates total health care delivery.

Official Definition

American Academy of Family Physicians

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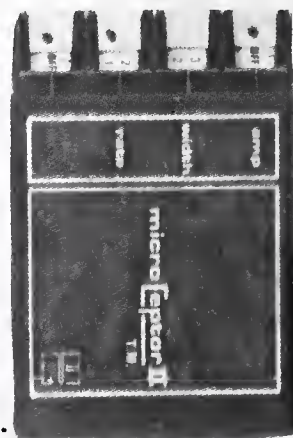
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This Is Your Medical Association

Spearfish doctor, **Forest Brady, M.D.**, has been named diplomate of the American Board of Family Practice.

* * * *

Aberdeen physician, **Charles L. Pelton, M.D.**, has joined the Physician Advisory Panel for Medical World News. The panel is made up of U.S. physicians who give the magazine suggestions on modern trends in medicine. Medical World News is published by McGraw-Hill of New York. Dr. Pelton specializes in family health services.

* * * *

Joe P. Chang, M.D., Aberdeen, has been elected president of St. Luke's Hospital medical-dental staff for 1981. Dr. Chang has been on active staff of St. Luke's for ten years. **Carlton Kom, M.D.** was elected vice president; **Tom Bunker, M.D.**, secretary-treasurer; **Barry Welge, M.D.**, medical section chairman; **Robert McGee, M.D.**, surgical section chairman; **Charles Pelton, M.D.**, primary care section chairman. Immediate past president is **Dr. Karl Kosse**.

* * * *

The Northeastern Mental Health Center of Aberdeen has hired **Terje Fokstuen, M.D.** as the medical director. Dr. Fokstuen comes to Aberdeen from Arvida, Sweden. He is a native of Norway and graduated from the Faculty of Medicine of the Univ. of Zurich, Switzerland. Dr. Fokstuen's wife and one son, 15, who is the youngest of three children, will be joining him in Aberdeen during the coming months.

* * * *

John J. Jacobsen, M.D., pediatrician has joined the Yankton Clinic, Yankton. After obtaining his degree in biological and ecological sciences at the U. of Nebraska Medical Center, Dr. Jacobsen went on to obtain his medical degree in 1977. He also completed his residency at the U. of Nebraska Medical Center in 1980.

Leonard M. Gutnik, M.D., Dept. of Internal Medicine, Central Plains Clinic, Sioux Falls, has recently become an Associate of the Society of Non-Invasive Vascular Technology. Dr. Gutnik is in charge of the peripheral vascular laboratory at the clinic.

* * * *

Eugene O. Hoxtell, M.D., Dept. of Dermatology, Central Plains Clinic, Sioux Falls, has recently passed the examination for certification of special competence in Dermatopathology.

* * * *

Central Plains Clinic, Ltd. of Sioux Falls, has announced that **Thomas M. Wilson, M.D.** has joined their Dept. of Pediatrics. Dr. Wilson is a Board Certified pediatrician who has been in practice in Somerset, Kentucky for the past several years. He and his wife, Jean, have five children.

* * * *

J. A. Muggly, M.D., Madison, has received the South Dakota Lung Association's highest honor, the Agnes M. Holdridge Award, for his contributions in the prevention and control of lung disease. He has served on the Lung Association's Board from 1960-69 and helped develop a system of tuberculosis control in South Dakota.

* * * *

Fred D. Leigh, M.D., 61, obstetrician and pediatrician in Huron, died recently. He had been with the Huron Clinic since 1948.

Dr. Leigh was born January 22, 1920 at Mt. Carroll, Ill. He graduated from the Univ. of Illinois Medical School. He served in the U.S. Navy during World War II. He was past president of the South Dakota Medical Association and the Huron Area Chamber of Commerce. He is survived by his wife, Helen and three children.

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This program has been reviewed and is acceptable for 15 prescribed hours by the American Academy of Family Physicians and 15 hours Category 1 AMA Physician Recognition Award.

WEDNESDAY, AUGUST 12, 1981

5:00 p.m. Board Meeting, SDAFP

THURSDAY, AUGUST 13, 1981

Morning Session

Wm. R. Tschetter, M.D., Moderator

8:00-9:00 Registration
9:00-9:10 Welcome—Wm. R. Tschetter, M.D.
9:15-9:55 George P. Henry, M.D.
Genetics for the Family Physician
10:00-10:15 Coffee, Conversation, Consultation
10:20-11:00 Paul A. Jensen, M.D.
Toxic Shock Syndrome
11:05-11:45 John A. Ochsner, M.D.
Vasectomy—Will It Hurt?
12:00-1:15 SDAFP Hosted Luncheon for Paid
Registrants and Spouses
Sam Nixon, M.D., President,
AAFP, Speaker

Afternoon Session

Raymond G. Nemer, M.D., Moderator

1:30-2:10 George P. Henry, M.D.
Genetic Amniocentesis
2:15-2:55 John A. Ochsner, M.D.
Circumcision, 1981
3:00-3:15 Coffee, Conversation, Consultation
3:20-4:00 Paul A. Jensen, M.D.
Conception Control, 1981
4:05-4:55 Panel Question and Answer Session
5:00 Meeting—Legislative Committee, South
Dakota Chapter, AAFP
Meeting—Education Committee, South
Dakota Chapter, AAFP

FRIDAY, AUGUST 14, 1981

Morning Session

Herbert Saloum, M.D., Moderator

8:00-9:00 Registration

9:00-9:40 Arnold H. Greenhouse, M.D.
The "Survey" Neurological Exam
9:45-10:00 Coffee, Conversation, Consultation
10:05-10:55 Richard E. Finlayson, M.D.
*Psychotherapy—Can the Family Physician
Be Effective?*
11:00-11:40 Sam Assam, M.D.
Intracranial Vascular Emergencies
11:45-1:15 SDAFP Hosted Buffet Luncheon for Paid
Registrants with a Speaker (select one)
Arnold Greenhouse, M.D.
Richard Finlayson, M.D.
Sam Assam, M.D.

Afternoon Session

Lawrence Finney, M.D., Moderator

1:20-2:00 Arnold H. Greenhouse, M.D.
TIA—Diagnosis and Management
2:05-2:45 Richard E. Finlayson, M.D.
Schizophrenia, 1981
2:50-3:30 Sam Assam, M.D.
Cervical Disc Syndrome
3:30-3:45 Coffee, Conversation, Consultation
3:45 Annual Business Meeting, South Dakota
Chapter, AAFP
6:30 Dinner in the Hills

SATURDAY, AUGUST 15, 1981

Morning Session

Charles L. Swanson, M.D., Moderator

8:00-9:00 Registration
9:00-9:30 Richard E. Finlayson, M.D.
Depression—Diagnosis
9:35-10:05 Arnold H. Greenhouse, M.D.
Use and Abuse of the CT Scan
10:05-10:15 Coffee, Conversation, Consultation
10:15-10:45 Sam Assam, M.D.
Extracranial Cerebrovascular Disease
10:50-11:20 Richard E. Finlayson, M.D.
Depression—Treatment
11:25-12:00 Panel Question and Answer Session

**MAKE PLANS TO ATTEND NOW, WRITE: BLACK HILLS SUMMER SEMINAR,
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S D

Future Meetings

July

Clinical Management of Coronary Disease and Exercise Testing, The Abbey Resort, Lake Geneva, WI, July 17-19. 13 hrs. AAFP & AMA Category I credit. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

Laboratory Design Seminar, San Diego, CA, July 22-24. Fee: \$400. Contact: Norman V. Steere & Assoc., Inc., 140 Melbourne Ave., SE, Minneapolis, MN 55414. Phone: (612) 378-2711.

Cardiac Rehabilitation, Orlando Hyatt, Orlando, FL, July 24-25. 13 hrs. AAFP & AMA Category I credit. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

Disorders of the Spine, Hyatt Regency, Minneapolis, MN, July 29-Aug. 1. 22 hrs. AMA Category I credits. Fee: \$350. Contact: CME, Box 293, Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

EKG Interpretation and Arrhythmia Management, Shanty Creek Hilton Resort, Bellaire, MI, July 31-Aug. 2. 13 hrs. AAFP & AMA Category I credit. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

August

Advances in Diagnostic Imaging, Hotel Scandinavia, Copenhagen, Denmark, Aug. 2-8. 30 hrs. AMA Category I credits. Contact: Dr. L. R. Muroff, Box 17241, Tampa, FL 33682. Phone: (813) 971-6000, ext. 297.

Great Debates in Otolaryngology, Washington Plaza Hotel, Seattle, WA, Aug. 6-8. 19.5 hrs. AAFP & AMA Category I credit. Fee: \$350. Contact: Registration, Cont. Med. Ed., SC-50, Univ. of Wash., Seattle, WA 98195. Phone: (206) 543-1050.

EKG Interpretation and Arrhythmias Management, Doubletree Inn, Monterey, CA, Aug. 7-8. 13 hrs. AAFP & AMA Category I credit. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

Arrhythmias and Cardiac Ischemia: Diagnosis & Management, Hyatt Regency, Montreal, Canada, Aug. 14-16. 13 hrs. AAFP & AMA Category I credits. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

The Sixth Annual Convention of the American College of International Physicians, Holiday Inn, Chicago, Ill., Aug. 20-23. 12 hrs. AMA Category I Credit. Contact: Am. College of International Phys., 3030 Lake Ave., Fort Wayne, IN 46805. Phone: (219) 424-7414.

Cardiac Rehabilitation, Sheraton Anaheim, Anaheim, CA, Aug. 21-22. 13 hrs. AAFP & AMA Category I credits. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

Recent Advances in Diabetes Management, Glacier Park Lodge, East Glacier, Mont., Aug. 29. 7-8 hrs. Category I credit. Contact: Stanlee Dull, Exec. Dir., Am. Diabetes Assoc., Box 2411, Great Falls, MT 59403. Phone: (406) 761-0908.

October

EKG Interpretation and Arrhythmia Management, Carousel Inn, Cincinnati, OH, Oct. 2-3. 13 hrs. AAFP & AMA Category I credits. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: 800-525-8646.

Laboratory Design Workshop, Minneapolis, MN, Oct. 5-9. Fee: \$600. Contact: Norman V. Steere & Assoc., Inc., 140 Melbourne Ave., SE, Minneapolis, MN 55414. Phone: (612) 378-2711.

The Sixth Annual International Body Imaging Conference, Hyatt Regency Hotel, Maui, Hawaii, Oct. 10-18. 25 hrs. Category I credit. Fee: \$365. Contact: Conference Secretary, Sixth Annual Internat'l. Body Imaging Conf., West Park Hosp., Dept. of Radiology, 22141 Roscoe Blvd., Canoga Park, CA 91304.

Cardiac Rehabilitation, Sheraton, Philadelphia, PA, Oct. 16-17. 13 hrs. AAFP & AMA Category I credits. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: 800-525-8646.

Complications in OB/GYN, Mayo Foundation Outreach Program, McKennan Hosp. Aud., Sioux Falls, SD, Oct. 23. 6 hrs. AAFP & AMA Category I credits. Contact: Ruth A. Muchow, Edu. Center, McKennan Hosp., 800 E. 21st St., Sioux Falls, SD. Phone: (605) 339-8000.

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Clinicopathological Conference
Seventy-Three Year Old Caucasian Female
With Abdominal Cramps And Diarrhea

Neonatal Resuscitation

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The FACT is that while most people can withstand normal, everyday anxieties, some people experience excessive and persistent levels of anxiety due to personal or clinical problems. An extensive national survey concluded that Americans who do use tranquilizers have substantial

Facts

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Seventy-Three Year Old Caucasian Female With Abdominal Cramps And Diarrhea

M. Rydberg, M.D.*
Discusser

J.F. Barlow, M.D.**
Editor

Case No. 877 501 7

This 73-year-old caucasian female was admitted to Sioux Valley Hospital with the chief complaint of abdominal cramps and foul smelling diarrhea of two weeks duration.

The patient had known atherosclerotic heart disease with angina pectoris on moderate exertion as well as diabetes mellitus controlled with 12-14 units of lente insulin daily and mild hypertension controlled by propranolol. She had difficulty for the past year with heartburn and had been treated with cimetidine for diagnoses of peptic esophagitis and gastroesophageal reflux. Several weeks prior to admission, she had gone on a trip to New England and two weeks prior to admission saw a physician because of abdominal soreness with cramping pain and foul smelling diarrhea with passage of small amounts of orange liquid stool without blood. There was a fair amount of tenesmus. Anorexia and weakness were progressive. There was very mild epigastric burning which radiated into the chest.

The patient had had a previous hysterectomy and bowel resection for obstructive bands. She had been observed several years prior to admission for possible myocardial infarction which was not documented. She had also had some vertigo attributed to labyrinthine dysfunction.

PHYSICAL EXAMINATION: Pulse: 80/min and regular; respirations: 20/min and regular; blood pressure 122 systolic and 70 diastolic; temperature 98.6°F. Examination of the neck and head was unremarkable. The chest was clear to auscultation and percussion. The heart was not enlarged and there was a regular rhythm with a Grade II (VI grades) apical systolic murmur. The abdomen revealed no tenderness or palpable

organs or masses. Pelvic examination was unremarkable. Neurological examination was within normal limits.

LABORATORY DATA: Urinalysis: slightly cloudy, specific gravity 1.011; pH 5.0, negative for protein, glucose, ketone bodies, bile and hemoglobin; sediment 3-6 white cells/hpf; 0-1 red cells/hpf. Hemoglobin 14.8 gm/dl, red count 4.65 million/mm³, ($4.65 \times 10^{12}/L$), hematocrit 46 vol/dl, normal red cell indices, total leukocyte count 4900/mm³ ($4.9 \times 10^9/L$), with a differential showing 21% segmented neutrophils, 5% monocytes and 64% lymphocytes, 10% of which were reactive. Sedimentation rate 72 mm/hr. Platelet count 198,000/mm³ ($19.8 \times 10^9/L$). Three stools for occult blood and ova and parasites were negative. Lactic dehydrogenase (LDH), alkaline phosphatase, aspartate aminotransferase, total bilirubin, total protein, calcium, phosphorus, glucose, blood urea nitrogen, creatinine, uric acid, and cholesterol were all within normal limits. The glucose did fluctuate between 130 and 200 mg/dl. A stool culture revealed campylobacter fetus subspecies jejuni. No other enteric pathogens were found.

DR. RYDERG: To summarize, this is an elderly diabetic, hypertensive woman with arteriosclerotic heart disease and angina pectoris. She had a sudden onset of foul smelling diarrhea with abdominal cramping and tenesmus following a trip to New England. She saw a physician for the above symptoms and passage of orange liquid stool without blood. We are not told if the stool was tested for occult blood. Because of progressive anorexia and weakness with persistence of the above symptoms, she was admitted to the hospital for evaluation.

There is no single definition of diarrhea but it may be defined as increased frequency (greater than

* Resident, Family and Community Medicine, Sioux Falls, SD.

**Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital, Sioux Falls, SD; Professor of Pathology, Department of Laboratory Medicine, School of Medicine, University of South Dakota.

three bowel movements per day), increased fluidity, (unformed, watery), or increased weight of stool (over 200 grams per day).²

The sudden onset and progressive nature of this woman's illness without similar prior episodes appears to place her illness in the diagnostic area of the acute diarrheas. She had had diabetes mellitus for an unspecified duration, but diabetic diarrhea is uncommon and usually presents with a watery diarrhea which is frequently nocturnal and may be associated with fecal or urinary incontinence. On physical examination, orthostatic hypotension with a visceral and peripheral neuropathy are present.¹ These were not noted in this case. Other possibilities from the history include intestinal ischemia secondary to arteriosclerosis. This is unlikely because this usually presents with a malabsorption syndrome and chronic diarrhea. Other possibilities, since she did have a bowel resection, are blind loop syndrome or short bowel syndrome. In blind loop syndrome the problem is a bacterial overgrowth in a diverted or blind loop. In short bowel syndrome, there is simply loss of absorptive surface due to the large amount of bowel resected. In both instances the diarrhea occurs after surgery.

What about the acute causes of diarrhea? In this patient these causes would be bacterial, viral, protozoal, or antibiotic associated. Diarrhea associated with antibiotics usually starts 4-9 days after starting the antibiotics. The etiology is thought to be alteration of bacterial flora, but there is only minimal evidence for this. The diarrhea is usually watery and mild.² Another form of antibiotic associated disease is characterized by pseudomembranous colitis linked to an enterotoxin which damages the intestinal mucosa causing an outpouring of fluid, mucus, and blood, and is produced by an anaerobe, *Clostridium difficile*.² In this patient, there is a possibility of an antibiotic related cause, even though medications were not mentioned since she did visit a physician. The evidence against this is absence of bloody stools and the apparent absence of antibiotic usage as well as the isolation of a potential gram negative pathogen.

Another cause of diarrhea could be protozoa such as *Entamoeba histolytica* or *Giardia lamblia* but three negative screens for ova and parasites tend to exclude these.

Viruses such as rotavirus, parvovirus, adenovirus, echovirus and the coxsackie viruses can also cause diarrhea. These are usually seen in children, but parvovirus and certain enteroviruses do affect adults. Viral gastroenteritis presents with vomiting and diarrhea usually severe lasting 36-48 hours. A mild persistent diarrhea may occur for several weeks but rarely develops into a malabsorption syn-

drome.^{1,3}

Finally, there are the bacterial causes of diarrhea. These can be divided into the mucosally invasive organisms and the noninvasive toxigenic organisms. *Shigella*, *Salmonella*, *Escherichia coli* and *Staphylococci* can be invasive organisms.^{1,2,3}

Shigellosis is characterized by fever, abdominal cramps and diarrhea. The incubation period is 1-2 days. Tenesmus is also present with nausea, vomiting, and myalgias being common. Stools are liquid and green in color with 1/5 to 1/3 of the patients having blood in the stools from superficial ulceration in the bowel mucosa. The more common organisms are *Shigella flexneri* and *Shigella sonnei*. These diseases are self limited and last about 7-10 days.

Salmonellosis is also an entity that presents with a persistent step-wise fever. Anorexia may develop with a rash (rose spots in typhoid fever). Also in typhoid, there is a leukopenia of 3000-4000/mm³ and a normochromic normocytic anemia. Constipation is an early symptom but diarrhea develops later in the disease. Salmonellosis can present as an acute gastroenteritis. Fever is a prominent sign and the abdominal pain can be quite severe.³

Escherichia coli can produce diarrhea via two mechanisms—one via enterotoxin which causes 50% of cases of travelers diarrhea and the second an enteroinvasive form which can ulcerate the bowel mucosa.

Staphylococci produce an enterotoxin mediated diarrhea from food poisoning that is abrupt in onset and relatively shortlived. An invasive staphylococcal antibiotic related pseudomembranous enterocolitis can also occur.

Vibrio cholera elaborates toxin which stimulates adenyl cyclase producing a severe watery painless diarrhea. *Vibrio parahaemolyticus* presents with nausea, vomiting, abdominal pain, diarrhea, and tenesmus 15-24 hours following ingestion of undercooked seafood such as shrimp or crab.³ In all the above causes of diarrhea, stool cultures provide the definitive diagnosis. Cultures for the above organisms were presumably negative except for campylobacter.

An older name for campylobacter fetus subspecies jejuni (CFssJ) was *Vibrio fetus*. In the 1970's campylobacter enteritis was finally recognized as causing human diseases although this had been suspected for approximately 30 years.⁴ Skirrow, in 1977, showed an incidence of approximately 7% of campylobacter as a cause of diarrhea in adults and children. Other studies reported a 6-34% incidence.^{1,6,8} In symptomless healthy adults and children, campylobacter is isolated in one percent or less of individuals.⁵

The epidemiology of campylobacteriosis is interesting. These organisms have been recognized as a major cause of animal disease resulting in abortions in sheep and cattle, and as a cause of diarrhea in pets.¹⁰ The transmission from animals to man is suspected but not proven. There have been several reports of isolation from cats, dogs, (specifically puppies), broiler chickens, and food implicated in animal to human transmission of this disease.^{11,12,13,14,15} At present, further confirmatory studies are needed.

The pathophysiology of campylobacteriosis is largely unclear at the present time. One article by Guerrant et al¹⁶ reviews 91 patients. The authors were unable to identify either heat stable or labile cholera-like enterotoxin, cytotoxicity, or invasiveness.¹⁶ In two reports, there was a suggestion of mucosal invasion as documented by rectal biopsy.^{18,19}

The incubation period is 3-5 days with a range of 1-10 days. The presentation is variable but usually within 24 hours with the acute onset of crampy abdominal pain, which incidentally can be severe enough to simulate an acute abdomen.²⁰ The diarrhea soon follows.^{16,17,18} This is initially watery but may progress to a mucoid character in about 50-60% of the cases. Blaser¹⁷ in a study of 514 people with diarrhea found bile stained diarrhea, blood or melena, and flatulence in approximately 50% of the cases.⁸ The fever usually peaks early and then declines later in the course of the disease.

Other symptoms include headache (60%) myalgias (50%), nausea (50%), arthralgias and backache (30%) and weight loss of greater than 2.3 kg (15%). Relapses occur in 20-30% of the cases reviewed.⁸

Laboratory features include stool leukocytosis in about 75% of patients, a peripheral blood leukocytosis of greater than 10,000/mm³ in about 60% and gross or occult stool blood in 50-60% of cases.⁶ Generally, hematocrit, electrolytes, glucose, and liver function tests are within normal limits.⁶ Blood cultures may or may not be positive.

Stool cultures are necessary for definitive diagnosis. These have to be plated on special selective media⁶ in a low oxygen atmosphere since campylobacter is microaerophilic, but not anaerobic.¹⁰

Recently, one study of 28 vibrio strains and three campylobacter strains were shown to be immobilized in distilled water, but were unaffected in Trypticase soy broth. This was presented as a possible rapid method for presumptive diagnosis.²¹

With the above clinical and laboratory data and positive stool culture, I believe this patient to have untreated or partially treated campylobacter enteritis.

Dr. Rydberg's Diagnosis Enteritis Due To Campylobacter Fetus Subspecies Jejuni

DR. BARLOW: In recent years there has been a renewed awareness that organisms of the genus campylobacter can produce serious human infections. Actually, only two species are commonly implicated-campylobacter fetus subspecies jejuni (CFssJ) which is a more frequent isolate from patients with self-limited diarrhea than shigella and salmonella, and campylobacter fetus subspecies intestinalis (CFssI) which may cause systemic infection in pregnant women, neonates, and immunosuppressed adults with a high mortality.

Originally known as vibrio fetus, the organism has been known as a serious cause of abortion in cattle since early in the century. Actually there are a variety of subspecies in the genus campylobacter (meaning curved rod) which have a variety of different characteristics.

The organism was separated from the vibrios in 1963. Although organisms of both the vibrio and campylobacter genera are both gram negative curved rods with a single polar flagellum producing darting motility and oxidase positivity, there are several basic differences including DNA homology (guanine + cytosine) and the fact that vibrios are facultatively anaerobic and produce acid from carbohydrates while campylobacter organisms are microaerophilic and neither ferment nor oxidize carbohydrates. For these reasons, campylobacter has been placed within the spirillaceae.

As I have said, CFssI can cause nonenteric bacteremias in pregnancy resulting in stillbirth or prematurity as well as pneumonia in the mother or meningitis and septicemia in neonates with a high mortality and septicemia in immunosuppressed or alcoholic adults. Thrombophlebitis, pericarditis, endocarditis, peritonitis, salpingitis, arthritis, and lung abscess have also been reported. Culture from blood may be accomplished but holding the cultures longer than usual may be necessary.

CFssJ is a cause of a usually self-limited but often severe gastroenteritis with ulceration of the ileum and colon. Crampy periumbilical abdominal pain, watery diarrhea accompanied with blood and pus, headache, fever, nausea, vomiting, malaise, and anorexia are common. The symptoms often but not always subside within a week. The disease can certainly mimic ulcerative colitis or Crohn's disease. This has been well described by Dr. Rydberg.

A large water-borne outbreak has occurred in Bennington, Vermont, involving 20% of 10,000 residents. Other smaller outbreaks in Colorado and in Toronto have been described. The disease has

been reported widely and may be transmitted via water, poultry, sick dogs and cats, unpasteurized milk, or person to person. It has also been seen in homosexual men. The CFssJ organism has been estimated to cause up to 5% of infectious diarrhea in this country and 3-11% of diarrhea in Europe. In one study 5% of 511 or 26 patients with diarrhea had the organisms isolated from the stool. 5 to 11 symptomatic household contacts but only one of 18 asymptomatic household contacts and none of 157 healthy persons yielded CFssJ in the stool. 17 of 20 affected persons tested had a fourfold rise of IgG antibodies. 78% had leukocytes and 60% had blood in the stool. In the same time period in which the 26 cases of CFssJ were detected, only 19 cases of salmonellosis, 13 cases of shigellosis, 2 cases of candidiasis, and 4 cases of giardiasis were detected as causes of infectious diarrhea. 1336 cases of CFssJ were reported in 9 months in England. In 1978, the peak incidence occurs at 1-5 years of age with highest rate of infection in the summer.

Isolation of CFssJ requires special conditions which include: 1) 42°-43°C incubation temperature, 2) microaerophilic atmosphere with 5-6% oxygen and 5-10% carbon dioxide, 3) special media. The temperature requirement demands a special incubator. There are now commercially available packets which create a suitable atmosphere for growth of CFssJ in bacteriologic jars. There are several media used. All incorporate various antimicrobial agents to inhibit other organisms. The best known is the media of Skirrow, which contains polymyxin, vancomycin, and trimethoprim and often amphotericin B in trypticase soy with 5% sheep blood. The colonies are moist gray nonhemolytic and appear like droplets of mucus. Our tact at this point has been to culture stool routinely for this pathogen. Several cases of CFssJ have already been isolated in this community.

*DR. PHILIP CARSON: How useful is the detection of fecal leukocytes in the investigation of patients with diarrhea? I have been told that it is useful in distinguishing mucosal colitis from ulcerative colitis or infectious diarrhea.

DR. BARLOW: I think fecal leukocytes indicate

that there is ulceration with inflammation within the gastrointestinal tract. This usually occurs in infectious diarrhea due to invasive organisms such as shigella, yersinia, campylobacter, invasive escherichia coli (E. coli) and sometimes salmonella. It is unlikely to occur in enterotoxin produced diarrheas such as from staphylococcal food poisoning or enterotoxin producing E. coli mediated diarrheas. Of course, ulcerative colitis might well have leukocytes in the stool.

**DR. R. D. SCHULTZ: I have read that the detection of fecal leukocytes is an excellent screening test. If they are present, this would indicate further workup with cultures trying to detect such organisms as campylobacter, yersinia, or shigella.

***DR. J. F. FOSS: I agree but one must remember the absence of fecal leukocytes does not rule out an invasive organism causing diarrhea. In one chart that was shown during this discussion only 11 of 14 of the patients with culture proved campylobacter diarrhea had fecal leukocytes.

****DR. JEAN ELLER: Would you comment on treatment of this disease?

DR. BARLOW: Erythromycin, gentamicin or tetracycline have all been employed successfully in treatment of diarrhea due to CFssJ.

FINAL DIAGNOSES ENTERITIS DUE TO CAMPYLOBACTER FETUS SUBSPECIES JEJUNI

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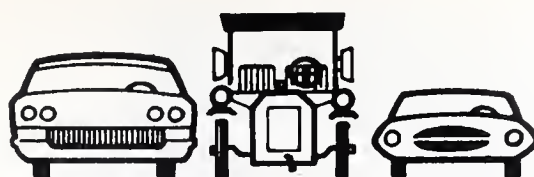
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Malpractice Crisis - Part II

Malpractice is back in the news again. The news is not good. Both New York and California physicians are facing higher liability premium rates in the near future. New York physicians are covered by two insurance carriers and one has requested a 71% increase on July 1, 1981 plus a projected 40% increase July 1, 1982 on top of a granted 24% increase last July! If the current request is granted, it would result in an average \$14,000 annual premium for the 18,000 New York physicians covered by this MD-owned medical malpractice insurance company. Dramatic increases in the cost of closing cases, plus increases in the number of new cases filed are the reason stated for the requested rate increases.

While South Dakota physicians are not experiencing these amazing rate increase requests from our carriers, it is ominously reminiscent of the mid-70's crisis years. Our new problem appears to be primarily the high cost of coverage. Solutions to the cost-crisis are difficult to out-line at this time. Additional legislative remedies may need to be proposed as our focus sharpens on specific areas of the problem.

Prevention must still be considered the most effective remedy for malpractice suits—just as it is for disease. Obviously the fewer cases filed, the lower the costs to all concerned. Adequate communication with patients is the area that deserves attention in any prevention program for physicians. Surveys continue to reveal that patients place a high priority on having respect, courtesy and concern shown to them by their doctors and other health care personnel. We need to remember that illness is an anxiety producing experience. Not only can this anxiety slow the process of healing but it can also prompt the displeased patient to consider a malpractice suit if a medical complication occurs.

According to the St. Paul Insurance Company Surveys, lack of patient rapport and physician-patient communications are the number one contributor to medical malpractice claims. Be sure your patients feel that you are a good listener and that you take time to answer their questions. St. Paul's survey reveals that our employees also need to reflect our concern for the patient's needs.



Dr. Robert Taylor, associate Professor of Family Medicine, Bowman Gray School of Medicine, writes in the February 1980 issue of "Physician's Management" that "every day physicians anger, alienate and lose patients because of failure to communicate". He feels it is largely the doctor's responsibility to promote good physician-patient interaction. This may help prevent noncompliance, anger and even litigation.

Openness to communication is an attitude. It is hard to learn and even more difficult to practice on busy days. The most useful way to improve physician-patient communication is for the physician to cultivate an attitude that says—"I am open to discussion. I will not ridicule or condemn you or become angry. As your physician, I am here to help and that can be best accomplished when we communicate freely". Improvement in our communication techniques is not the complete answer to the malpractice problem of today. However, it is at the very heart of any malpractice prevention program. If you don't have patient rapport before a complication, how can you expect to develop it afterwards?

Please share with me any suggestion you have concerning this important concern. If further legislation is felt to be part of the solution, the SDSMA will be ready to develop a specific legislative package for your review.

Very sincerely yours,

Bruce Lushbough

Bruce Lushbough, M.D., President
South Dakota State Medical Association

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EQUAGESIC—Abbreviated Summary

INDICATIONS: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Possibly effective for the treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease or tension headache. Final classification of the less-than-effective indications requires further investigation.

The effectiveness of Equagesic in long-term use, i.e. more than four months, has not been assessed by systematic clinical studies. The physician should periodically reassess usefulness of the drug for the individual patient.

CONTRAINDICATIONS: Equagesic should not be given to individuals with a history of sensitivity or severe intolerance to aspirin, meprobamate, or ethoheptazine citrate.

WARNINGS: Careful supervision of dose and amounts prescribed for patients is advised, especially with those patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g. alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on or habituation to the drug. Where excessive dosage has continued for weeks or months, dosage should be reduced gradually rather than abruptly stopped, since withdrawal of a "crutch" may precipitate withdrawal reaction of greater proportions than that for which the drug was originally prescribed. Abrupt discontinuance of doses in excess of the recommended dose has resulted in some cases in the occurrence of epileptiform seizures.

Special care should be taken to warn patients taking meprobamate that tolerance to alcohol may be lowered with resultant slowing of reaction time and impairment of judgment and coordination.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with the use

of minor tranquilizers (meprobamate, chlordiazepoxide, and diazepam) during the first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physicians about the desirability of discontinuing the drug.

Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breast-feeding patients, the drug's higher concentration in breast milk as compared to maternal plasma levels should be considered.

Preparations containing aspirin should be kept out of the reach of children. Equagesic is not recommended for patients 12 years of age and under.

PRECAUTIONS: Should drowsiness, ataxia, or visual disturbance occur, the dose should be reduced. If symptoms continue, patients should not operate a motor vehicle or any dangerous machinery. Suicidal attempts with meprobamate have resulted in coma, shock, vasomotor and respiratory collapse, and anuria. Very few suicidal attempts were fatal, although some patients ingested very large amounts of the drug (20 to 40 gm). These doses are much greater than recommended. The drug should be given cautiously, and in small amounts, to patients who have suicidal tendencies. In cases where excessive doses have been taken, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Hyperventilation has been reported occasionally. Any drug remaining in the stomach should be removed and symptomatic treatment given. Should respiration become very shallow and slow, CNS stimulants (e.g., caffeine, Metrazol, or amphet-

mine, may be cautiously administered. If severe hypotension develops, pressor amines should be used parenterally to restore blood pressure to normal levels.

ADVERSE REACTIONS: A small percentage of patients may experience nausea with or without vomiting and epigastric distress. Dizziness occurs rarely when meprobamate and ethoheptazine citrate with aspirin is administered in recommended dosage. The meprobamate may cause drowsiness but, as a rule, this disappears as therapy is continued. Should drowsiness persist and be associated with ataxia, this symptom can usually be controlled by decreasing the dose, but occasionally it may be desirable to administer central stimulants such as amphetamine or mephentermine sulfate concomitantly to control drowsiness.

A clearly related side effect to the administration of meprobamate is the rare occurrence of allergic or idiosyncratic reactions. This response develops, as a rule, in patients who have had only 1-4 doses of meprobamate and have not had a previous contact with the drug. Previous history of allergy may or may not be related to the incidence of reactions.

Mild reactions are characterized by an itchy urticarial or erythematous, maculopapular rash which may be generalized or confined to the groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema and fever have also been reported.

More severe cases, observed only very rarely, may also have other allergic responses, including fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case), and hyperthermia. Treatment should be symptomatic such as administration of epinephrine, antihistamine, and possibly hydrocortisone. Meprobamate should be stopped, and reinstitution of therapy should not be attempted.

Rare cases have been reported where patients receiving meprobamate suffered from aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia. In nearly every instance reported, other toxic agents known to have caused these conditions have been associated with meprobamate. A few cases of leukopenia during

continuous administration of meprobamate are reported, most of these returned to normal without discontinuation of the drug. Impairment of accommodation and visual acuity has been reported rarely.

OVERDOSE: Two instances of accidental or intentional significant overdose with ethoheptazine citrate combined with aspirin have been reported. These were accompanied by symptoms of CNS depression, including drowsiness and light-headedness, with uneventful recovery. However, on the basis of pharmacological data, it may be anticipated that CNS stimulation could occur. Other anticipated symptoms would include nausea and vomiting. Appropriate therapy of signs and symptoms as they appear is the only recommendation possible at this time. Overdosage with ethoheptazine combined with aspirin would probably produce the usual symptoms and signs of salicylate intoxication. Observation and treatment should include induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions.

DESCRIPTION: Each Equagesic tablet contains 150 mg meprobamate, 75 mg ethoheptazine citrate and 250 mg aspirin.

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*This drug has been evaluated as possibly effective for this indication.

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WYGESIC—Abbreviated Summary

INDICATION: For the relief of mild-to-moderate pain.

CONTRAINDICATION: Hypersensitivity to propoxyphene or to acetaminophen.

WARNINGS: CNS ADDITIVE EFFECTS AND OVERDOSE: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, or other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. Toxic effects and fatalities have occurred following overdoses of propoxyphene alone or in combination with other CNS depressants. Most of these patients had histories of emotional disturbances or suicidal ideation or attempts, as well as misuse of tranquilizers, alcohol, or other CNS-active drugs. Caution should be exercised in prescribing large amounts of propoxyphene for such patients (see Management of Overdosage).

DRUG DEPENDENCE: Propoxyphene can produce drug dependence characterized by psychic dependence and less frequently physical dependence and tolerance. It will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics. The abuse liability of propoxyphene is qualitatively similar to codeine's although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine.

USAGE IN AMBULATORY PATIENTS: Propoxyphene may impair the mental and/or physical abilities required for potentially hazardous tasks, e.g. driving a car or operating machinery. Patients should be cautioned accordingly.

USAGE IN PREGNANCY: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. **INSTANCES OF WITHDRAWAL SYMPTOMS IN THE NEONATE HAVE BEEN REPORTED FOLLOWING USAGE DURING PREGNANCY.** Therefore, propoxyphene should not be used in pregnant women unless, in the

judgement of the physician, the potential benefits outweigh the possible hazards.

USAGE IN CHILDREN: Propoxyphene is not recommended for children because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric group.

PRECAUTIONS: Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine. The CNS depressant effect of propoxyphene may be additive with other CNS depressants, including alcohol.

ADVERSE REACTIONS: The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting. These seem more prominent in ambulatory than in nonambulatory patients, some of these reactions may be alleviated if the patient lies down.

Other adverse reactions include constipation, abdominal pain, skin rashes, light-headedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances. The chronic ingestion of propoxyphene in doses over 800 mg per day has caused toxic psychoses and convulsions. Cases of liver dysfunction have been reported.

DRUG INTERACTIONS: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, and other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended (see Warnings). Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine.

MANAGEMENT OF OVERDOSAGE: SYMPTOMS: The manifestations of serious overdosage with propoxyphene are similar to those of narcotic overdosage and include respiratory depression (a decrease in respiratory rate and/or tidal volume), Cheyne-Stokes respiration, cyanosis, extreme somnolence progressing to stupor or coma, pupillary constriction, and circulatory collapse. In addition to these characteristics, which are reversed by narcotic antago-

nists such as naloxone, there may be other effects. Overdoses of propoxyphene can cause delay of cardiac conduction as well as focal or generalized convulsions, a prominent feature in most cases of severe poisoning. Cardiac arrhythmias and pulmonary edema have occasionally been reported, and apnea, cardiac arrest, and death have occurred.

Symptoms of massive overdosage with acetaminophen may include nausea, vomiting, anorexia, and abdominal pain, beginning shortly after ingestion and lasting for 12 to 24 hours. However, early recognition may be difficult since early symptoms may be mild and nonspecific. Evidence of liver damage is usually delayed. After the initial symptoms, the patient may feel less ill, however, laboratory determinations are likely to show a rapid rise in liver enzymes and bilirubin. In case of serious hepatotoxicity, jaundice, coagulation defects, hypoglycemia, encephalopathy, coma, and death may follow. Renal failure due to tubular necrosis and myocardialopathy, have also been reported.

Ingestion of 10 grams or more of acetaminophen may produce hepatotoxicity. A 13-gram dose has reportedly been fatal.

TREATMENT: Primary attention should be given to the reestablishment of adequate respiratory exchange through provision of a patent airway and institution of assisted or controlled ventilation. The narcotic antagonists, naloxone, nalmorphine, and levallorphan, are specific antidotes against the respiratory depression produced by propoxyphene. An appropriate dose of one of these antagonists should be administered preferably I.V., simultaneously with efforts at respiratory resuscitation and the antagonist should be repeated as necessary until the patient's condition remains satisfactory. In addition to a narcotic antagonist, the patient may require careful titration with an anticonvulsant to control seizures. Analeptic drugs (e.g. caffeine or amphetamine) should not be used because of their tendency to precipitate convulsions.

Oxygen, IV fluids, vasopressors and other supportive measures should be used as indicated. Gastric lavage may be helpful. Activated charcoal can absorb a significant amount of ingested propoxyphene. Dialysis is of little value in poisoning by propoxyphene alone. Acetaminophen is rapidly absorbed, and efforts to remove the drug from the body should not be delayed. Copious gastric lavage and/or induction of emesis may be indicated. Activated charcoal is probably ineffective unless administered almost immediately after acetaminophen ingestion. Neither forced diuresis nor hemodialysis appears to be effective in removing acetaminophen. Since acetaminophen in overdose may have an antidiuretic effect and may produce renal damage, administration of fluids should be carefully monitored to avoid overload. It has been reported that mercaptamine (cysteine) or other thiol compounds may protect against liver damage if given soon after overdosage (8-10 hours). N-acetylcysteine is under investigation as a less toxic alternative to mercaptamine, which may cause anorexia, nausea, vomiting and drowsiness. Appropriate literature should be consulted for further information (JAMA 237:2406-2407, 1977). Clinical and laboratory evidence of hepatotoxicity may be delayed up to one week. Acetaminophen plasma levels and half-life may be useful in assessing the likelihood of hepatotoxicity. Serial hepatic enzyme determinations are also recommended.

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14

Neonatal Resuscitation

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ABSTRACT

The transition from fetal to neonatal life involves complex respiratory, circulatory and metabolic changes. Therefore, it is not surprising that a physician who performs deliveries will occasionally encounter a neonate in need of assistance. The approach to a sick neonate should first include clearing of the airway, oxygen administration, and if necessary, ventilatory support with a resuscitation bag and mask or endotracheal tube. Cardiac activity must be monitored and external massage initiated if the heart rate remains below

100 bpm with ventilatory support. In addition to these procedures, one must maintain the infant's thermoneutral environment by drying the skin and placing the infant under a radiant heat source. Since premature and distressed infants metabolize their glycogen stores rapidly, all sick neonates should be screened for hypoglycemia. Blood gas analysis and a hematocrit may also be valuable in the assessment of the distressed neonate. Aside from knowing how to resuscitate a baby, it is imperative that every delivery room is fully equipped and well staffed so that emergency stabilization may be performed promptly and efficiently.

Any physician performing deliveries, will at some time be faced with an infant in need of resuscitation. The advent of modern neonatal medicine has permitted the survival of neonates with medical disorders which were often fatal in the past. The sophistication of modern intensive care nurseries is of little value if the newborn is permitted to sustain irreparable damage during the first minutes of life. It is in

the interest of eliminating such perinatal insult that the following material is presented.

Physiology of Transition to Extrauterine Life

The immediate neonatal period is of great importance, because of the multiple and complex physiologic adaptations which occur. First, consider the perinatal circulatory dynamics of the infant. The cardiac circulation in the human fetus is significantly different from the adult. The right ventricle of the fetus accounts for two-thirds of the total ventricular output, while the left ventricle accounts for only one-third. Well oxygenated blood returning to the heart, from the inferior vena cava and placental cir-

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culation, is directed across the foramen ovale through the left heart; whereas, the remainder of the venous return is shunted through the ductus arteriosus into the descending aorta (Figure 1 top). Approximately 7% of the cardiac output circulates through the lungs in utero, because of the very high pulmonary arterial resistance in the fetus. Following delivery, the large volume of blood returned to the right heart from the placental circulation is diminished, the pulmonary vascular resistance diminishes with the initiation of ventilation, and the systemic vascular resistance increases. With increased pulmonary blood flow, the pressure in the left heart increases, obliterating the right-to-left shunting of blood through the ductus arteriosus and foramen ovale (Figure 1 bottom). Each of three factors, including hypoxia, acidosis and cold stress may increase the pulmonary vascular resistance and decrease the pulmonary blood flow during the first two weeks of neonatal life. The combination of hypoxia and acidosis may reduce pulmonary blood flow more than either factor alone. Such persistence of the fetal circulatory pattern in the neonate may be responsible for severe and refractory hypoxemia.

Next, consider the respiratory changes at the time of parturition. The fetal lung contains an ultrafiltrate of plasma, which is not amniotic fluid. At delivery, the vagina and pelvic floor squeeze the fetal chest, forcing two-thirds of the fluid from the lungs; the remainder is removed after birth by the lymphatic and capillary systems. Small premature infants, and those born by Cesarean section, do not go through this transition, possibly accounting for their increased difficulty in establishing respiration.

Most infants begin breathing within thirty seconds, and establish rhythmic respiration by ninety seconds after birth. The outward recoil of the chest wall following vaginal delivery fills the lungs with air. The brain stem respiratory centers then stimulate rhythmic respiration. Many additional factors may stimulate ventilation including: pain, cold, touch, noise, cord clamping, acidosis, hypoxia and hypercarbia. Severe acidosis, hypoxia, maternal drugs (e.g., narcotics) and local anesthetics depress respiration.

Experimental animals, subjected to total neonatal asphyxia, demonstrate two phases of apnea during the first ten minutes of hypoxia. Initially, rapid gasps occur which are accompanied by muscular effort and thrashing movements of extremities. This ceases after one minute, the heart rate drops to approximately 100 bpm and primary apnea occurs. During primary apnea, spontaneous respiration can be stimulated by appropriate manipulation or ventilatory support. If allowed to progress, a series of spontaneous deep gases follow for five minutes. These be-

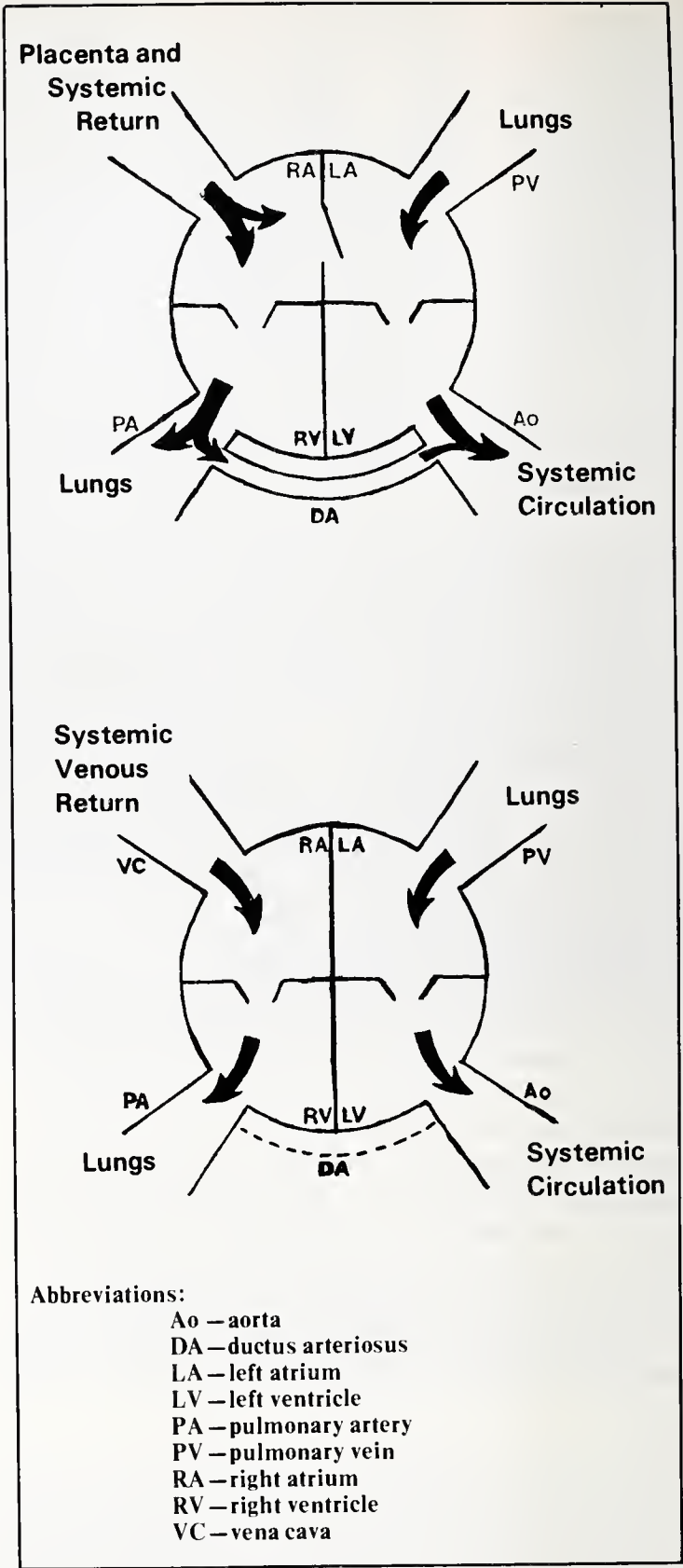


Figure 1
 Schematic diagram of the intracardiac circulation in the fetus (Top) and the neonate (Bottom).

come gradually weaker and terminate in secondary apnea at 8 minutes of asphyxia. At this time, spontaneous respiration cannot be induced and death occurs within several minutes. The longer the delay in initiating resuscitation, the longer the period of re-

suscitation necessary to recover spontaneous ventilation.

It is important for the clinician to recognize these physiologic changes so that he may recognize the stages of asphyxia, and more effectively correct pathologic imbalances during transition. It is important to realize that there are maternal, intrapartum, and fetal disorders which may be responsible for severe neonatal depression (Table I).

Table I
Factors Associated With
Neonatal Asphyxia

Maternal
Medical disorders
Anemia
Malnutrition
Drug Abuse
Age less than 16 or greater than 35 years
History of previous perinatal morbidity or mortality
History of previous infant with a hereditary disorder
Poor prenatal care
Amniotic membranes ruptured for over 24 hours
Infection
Antepartum Hemorrhage
Falling Estriol Values
Blood type or group isoimmunization
Eclampsia/Pre-eclampsia
Surgery during pregnancy
Intrapartum
Cephalopelvic disproportion
Abnormal fetal presentation
Cesarean section
Precipitous or difficult delivery
Forceps delivery
Prolonged labor
Maternal hypotension
Cord compression or prolapse
Analgesic or sedative drugs within two hours of delivery
Fetal
Multiple gestation
Poly-/Oligohydramnios
Immature Lecithin/Sphingomyelin Ratio
Premature or postmature delivery
Abnormalities of intrauterine growth (LGA, SGA)
Meconium staining
Fetal acidosis
Abnormalities of the fetal heart tones

Table II
Apgar Score

Sign	0	1	2
Heart rate	Absent	< 100	> 100
Respiratory effort	Absent	Slow, irregular	Good, crying
Muscle tone	Flaccid	Some flexion of extremities	Active motion
Reflex irritability	No response	Grimace	Cough and sneezing
Color	Blue, pale	Acrocyanosis	Totally pink

Table III
Resuscitation Equipment

Overhead radiant warmer.
Heated, humidified oxygen.
Wall suction and sterile catheters.
Oral airway.
Suction bulb.
Laryngoscope with size 0 and 1 straight blades.
Endotracheal tubes, sizes 2.5, 3.0 and 3.5 mm
Umbilical vessel catheterization tray with 3.5 and 5 Fr. umbilical catheters.
Glucose oxidase test strips (Dextrostix)

Resuscitation

In 1953, Virginia Apgar described a scoring system for neonates in an attempt to assess the need for resuscitation. This system assigned a score of either 0, 1 or 2 points for heart rate, respiratory effort, muscle tone, reflex irritability and color of a newly delivered infant at one and five minutes of age (Table II). A total score of 0 to 3 was consistent with a severely depressed infant requiring immediate resuscitation; whereas, a score of 4 to 7 correlated with a moderately depressed infant, and a score of 7 to 10 with a nondepressed infant. Although this scoring system has been useful in evaluating the amount of asphyxia which a newborn has sustained, one minute is too long to wait to make the decision to perform resuscitation.

Every delivery suite should be equipped with the items necessary for neonatal resuscitation (Table III). This equipment should be conveniently located and clearly displayed so that it is instantaneously available at the time of need. One individual in the delivery room should specifically be responsible for the care of the newborn. This may be a nurse or physician; however, if complications of any kind are anticipated, this individual must be familiar with all aspects of neonatal resuscitation.

A suggested outline for resuscitation is presented in Figure 2. At the time of delivery, the nose and mouth should be suctioned gently with a bulb syringe, and the infant placed under a radiant warmer with the head tilted down. The infant should be thoroughly dried and a 15 second examination performed to determine the need for resuscitation. This examination includes an assessment of the heart rate, respiratory effort, color and muscular activity. Under normal circumstances, spontaneous respiration will begin with the stimulation from drying. It is important to avoid cold stress by keeping the child in a warm environment (34.5-35° C.). It may be

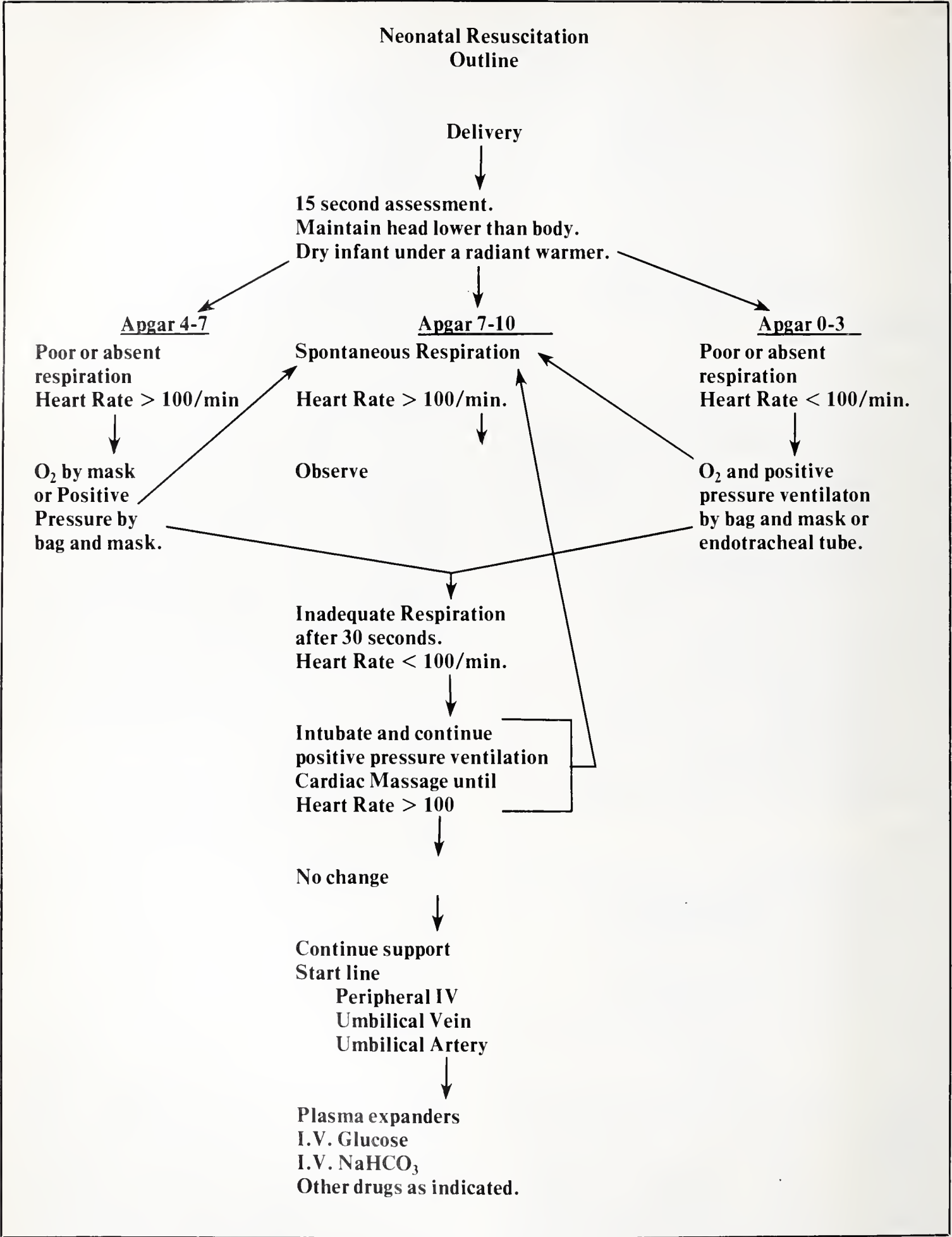


Figure 2

necessary to administer oxygen for a short period to relieve cyanosis. In the instance where the child has respiratory depression, the initial management should consist of positive pressure ventilation with a resuscitation bag, mask and 100% oxygen. Usually, ventilation can be maintained in this way. It is important to constantly monitor the infant's heart rate; if it remains below 100 bpm with respiratory support, external cardiac massage should be instituted. If ventilation cannot be maintained with a bag and mask, or if it becomes evident that long term ventilatory support is necessary, an oral or nasal tracheal tube should be inserted. In most instances, endotracheal intubation should be performed under closely monitored conditions and it should not jeopardize the infant's condition.

If positive pressure ventilation is required, it should be monitored by observing the movement of the infant's chest wall. The respiratory rate should be maintained at 40 breaths/min., with pressure applied so as to gently move the chest wall. In an infant who has not yet taken a breath, over 40 cm H₂O pressure may be necessary to expand the lungs, compared to a normal ventilatory pressure of 14 to 16 cm H₂O. Infants who are premature or have pulmonary disease, usually have poor lung compliance and require greater pressures to maintain gentle chest wall movement. This is important because most of the available resuscitation bags are equipped with a "pop-off valve" so that only 30 cm H₂O pressure can be administered. In a distressed infant, it may be necessary to occlude this valve so that adequate pressure may be delivered. It must be remembered, that too much pressure can produce adverse effects such as pulmonary interstitial emphysema (perivascular air dissection) or pneumothoracies.

When the heart rate remains less than 100 bpm and does not increase with sixty seconds of assisted ventilation, external cardiac massage should be initiated. This is performed by an assistant who places two fingers over the heart just to the left of the lower sternal border. Pressure is applied for a 1 to 2 cm depression in the chest wall. The rate of massage should be 100 to 120 bpm and synchronized so that ventilation at 40 breaths/min. can be administered (1:3 ratio). Cardiac massage may be stopped periodically to assess improvement.

In most instances, it is possible to obtain an adequate response with the use of external cardiac massage and assisted ventilation; however, if vigorous supportive therapy has been administered for three minutes without a response, chemical management should be considered. Any route of access to the circulatory system is acceptable, including a peripheral intravenous, an umbilical venous or an umbili-

cal arterial line. In an acute emergency, the most expedient procedure is to insert an umbilical venous catheter. If possible, this catheter should be passed through the ductus venosus into the inferior vena cava (10-12 cm) so that sclerosing agents such as hypertonic sodium bicarbonate or glucose solutions are not injected directly into the portal venous system. If the catheter cannot be passed into the vena cava, the portal system should be avoided by anchoring the catheter superficially at a depth of 4-5 cm. The drugs commonly used in neonatal resuscitation are listed, with their doses, in Table IV.

Hypotension in the neonate may be recognized by pallor, mottling of the skin, poor capillary filling, weak or thready pulses, or low blood pressure as measured by ultrasound or with an umbilical arterial catheter. In addition to being hypotensive on the basis of severe asphyxia, acute or chronic blood loss may produce shock in the neonate. A perinatal history of abruptio placenta, placenta previa or maternal bleeding suggests that the neonate may also have experienced blood loss. In such cases, an early hematocrit can be of value. Hypotensive infants should receive immediate plasma expansion in the form of whole blood or a 5% protein solution (Plasmanate, 5% salt poor albumin), given intravenously over 10 minutes in dosages of 10-20 ml/kg. Cord blood, sterilely obtained in a heparinized syringe at delivery, may also be used for volume expansion.

Stressed and premature infants rapidly exhaust their glycogen stores and are subject to hypoglycemia (whole blood or serum glucose < 20 mg% in a premature or < 30% in a full term infant). To

Table IV
Drugs for Resuscitation

Drug	Indication	Dosage
Whole blood or 5% protein solution	Blood loss, shock or hypotension	10-20 ml/kg IV
D10-12.5W	Hypoglycemia	2-4 ml/kg IV
Naloxone	Neonatal depression secondary to maternal narcotic administration	0.01 mg/kg IV
NaHCO ₃ (1 mEq/ml)	Metabolic acidemia or asphyxia not responsive to resuscitation	1 mEq/kg, diluted 1:2 with sterile water over 10-15 min. IV
Epinephrine 1:10,000 solution	Cardiac arrest or bradycardia non-responsive to resuscitation	0.1 ml/kg IV
Calcium Gluconate 10%	Bradycardia	1.0 ml/kg IV
Calcium Chloride 10%	Bradycardia	0.1 ml/kg IV

avoid such complications, sick neonates should receive a 10% glucose solution intravenously. In severely depressed or symptomatic (e.g., jittery, hypotonic, seizing) infants, it may be necessary to administer 1-2 ml/kg of D₂₅W solution by slow intravenous infusion. If the infant is not having symptomatic hypoglycemia, the authors usually prefer to administer a dilute glucose solution (D₁₀W, D_{12.5}W), because of the potential complications of hypertonic solutions (e.g., liver damage, skin necrosis). All stressed neonates should have their blood sugar checked during the first hour of life and hourly thereafter until it remains consistently above 45 mg%.

Nearly all asphyxiated infants have a combined respiratory and metabolic acidosis. The respiratory acidosis can be alleviated by positive pressure ventilation. The metabolic acidosis is caused by the accumulation of lactic acid from anaerobic metabolism during episodes of hypotension and hypoxia. It is important to correct this by the administration of buffer, usually sodium bicarbonate. In cases of a severe or prolonged asphyxia, 1-2 mEq/kg of sodium bicarbonate diluted to 0.5 mEq/ml with sterile water is given by slow intravenous infusion over 10-15 minutes. Subsequent bicarbonate therapy should be administered more precisely based upon blood gases.

In assessing oxygenation and the acid-base status of a neonate, it is important to perform arterial blood gases. The skin color is an unreliable index of oxygenation because fetal hemoglobin binds oxygen more closely than adult hemoglobin; therefore, cyanosis occurs at a P_aO₂ of 30 to 40 mmHg in infants as opposed to 40 to 50 mmHg as seen in adults. Capillary blood gases yield accurate pH and PCO₂ values but the PO₂ is quite variable. Optimally, a radial arterial blood gas should be performed on all sick neonates, but a capillary gas may be useful for determining acid-base balance, and the effectiveness of ventilation, if an arterial sample cannot be obtained. Transcutaneous monitoring of the PO₂ may be useful; however, the transcutaneous PO₂ must be standardized against the arterial PO₂ to be reliable.

Another pharmacologic agent commonly used in neonatal resuscitation is the narcotic antagonist naloxone. The indication for this drug is maternal narcotic administration with subsequent neonatal respiratory depression. The time for the peak concentration of transplacentally acquired narcotics in the fetus is 2 hours. Delivery of an infant at this time would predispose him to maximal depression on this basis. Currently, naloxone, available in a pediatric dose vial, is preferred over nalorphine because the latter is a potential respiratory depressant.

A 1:10,000 solution of aqueous epinephrine may

be indicated for cardiac arrest, or severe bradycardia, not responsive to routine resuscitative measures. This drug is administered intravenously or by intracardiac injection; however, if given intravenously, it is necessary to continue cardiac massage to hasten its pharmacologic effect. The standard dose of epinephrine is 0.1 to 0.3 ml/kg given after sodium bicarbonate. Calcium gluconate or calcium chloride may be administered in cases where severe bradycardia persists despite the previously outlined therapeutic measures.

Special Considerations

There are a number of clinical problems in which the approach to the patient must differ from the previous outline. The first of these is meconium staining. Meconium aspiration carries a 20 to 50% mortality rate; however, with proper management, it is almost entirely preventable. In cases where the amniotic fluid is slightly discolored by meconium, but gross meconium is not present, special management is not necessary. If gross meconium is noted at the time of delivery, the following regime is suggested:

1. After delivering the infant's head, but before delivering the shoulders, the nose, mouth and pharynx should be thoroughly suctioned with a DeLee suction catheter.
2. Immediately after the delivery of the infant, repeat suctioning of the upper airway should be performed while the infant is being dried and placed under a radiant warmer.
3. The trachea should be visualized with a laryngoscope and meconium aspirated by direct suctioning through an endotracheal tube. Suctioning is repeated until no more meconium is present.
4. Thereafter, the infant may be ventilated and resuscitated with positive pressure as indicated.
5. The gastric contents should be aspirated prior to releasing the infant to the nursery.

It is imperative that the trachea is cleared before spontaneous or positive pressure ventilation begins. Failure to do this may disseminate meconium throughout the airways producing the diffuse chemical pneumonitis of the meconium aspiration syndrome.

The second situation in which the clinician's approach to resuscitation must be modified is that of congenital diaphragmatic hernia. In these cases, the infant may present with profound respiratory distress and cyanosis in the delivery room, or within the first few hours of life. Clinical findings suggestive of congenital diaphragmatic hernia include:

1. Unilaterally decreased breath sounds (usually, but not always, on the left side).

2. A scaphoid abdomen.
3. The radiographic finding of bowel gas in the hemithorax (usually on the left side).

Although diaphragmatic hernias are uncommon, it is imperative that they are recognized early so that positive pressure by mask is not administered. Such treatment causes gastric distension and increased bowel gas producing profound respiratory compromise as the bowel in the hemithorax becomes dilated. Appropriate management in these cases includes constant gastric suction and endotracheal intubation. Immediate referral to a tertiary neonatal intensive care unit with surgical facilities is imperative.

The last special situation, to be discussed, is the occurrence of a spontaneous pneumothorax. Any neonate, who acutely becomes cyanotic and hypotensive with respiratory distress, should be evaluated for a pneumothorax. It is important to recognize that this may occur in the full term, as well as the small premature infant with respiratory distress. Infants, in whom positive pressure ventilation has been administered, are at particular risk for pneumothoraces. Physical examination of these infants may reveal unilaterally depressed breath sounds, shift of the heart sounds, positive transillumination of the hemithorax, and the finding of a unilaterally increased lucency on the chest x-ray often with a distinct pleural line and lung collapse. Acute management of this disorder includes needle aspiration with a 23 gauge butterfly needle attached to a three-way stopcock and a 50 cc syringe. The needle is inserted directly over the 4th rib in the mid-clavicular line. As the needle is inserted, an assistant aspirates until air is withdrawn. With relief of the pneumothorax, the infant's clinical condition will dramatically improve. Aspiration is continued until no more air can be aspirated, or until a chest tube can be inserted. Respiratory and circulatory support must be maintained throughout this procedure.

Conclusion

Care of the resuscitated infant does not end after resuscitation. Continued close monitoring of the temperature, respiratory rate, heart rate, blood pressure, glucose, P_aO_2 , P_aCO_2 , pH and hematocrit is required in every infant who was sequelae which can occur after periods of significant perinatal stress. Many of the infants, who continue to have difficulty, may be candidates for referral to tertiary neonatal intensive care units. Immediate, vigorous neonatal resuscitation change dramatically the physical and neurologic prognosis of the severely depressed neonate.

(Continued on page 22)

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Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg; bismuth subgallate, 22.5 mg; bismuth resorcin compound, 17.5 mg; benzyl benzoate, 12.0 mg; Peruvian balsam, 18.0 mg; zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water.

Anusol-HC Suppositories and Anusol-HC Cream help to relieve pain, itching and discomfort arising from irritated anorectal tissues. These preparations have a soothing, lubricant action on mucous membranes, and the antiinflammatory action of hydrocortisone acetate in Anusol-HC helps to reduce hyperemia and swelling.

The hydrocortisone acetate in Anusol-HC is primarily effective because of its antiinflammatory, antipruritic and vasoconstrictive actions.

Indications and Usage: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain, itching and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas, pruritus ani and relief of local pain and discomfort following anorectal surgery.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

Contraindications: Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

Precautions: General: Symptomatic relief should not delay definitive diagnoses or treatment.

Prolonged or excessive use of corticosteroids might produce systemic effects.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Anusol-HC is not for ophthalmic use.

Pregnancy

See "WARNINGS"

Pediatric Use

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

Dosage and Administration: Anusol-HC Suppositories—

Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain comfort with regular Anusol Suppositories.

Anusol-HC Cream—Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

How Supplied: Anusol-HC Suppositories—boxes of 12

(N 0071-1089-07) and boxes of 24 (N 0071-1089-13) in silver foil strips with Anusol-HC printed in black.

Anusol-HC Cream—one-ounce tube (N 0071-3090-13) with plastic applicator.

Store between 59°-86°F (15°-30°C).
1089G010

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**S
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Letters To The Editor

I would like to thank the Association for the Award I received for the coming academic year. As you are well aware, the Association's support of the USD School of Medicine is greatly appreciated, and his personal support is also appreciated as much as it is economically beneficial. Thank you for helping me achieve my goal of a career in medicine.

Calvin Sprik
415 W. Cedar St.
Vermillion, SD 57069

I wish to point out serious errors in the article entitled "Myxedema Coma" by Timothy Lamphier in the April 1981 issue of the Journal. On page 15 and 17 the dosage for Cytomel (liothyronine) is listed incorrectly. The correct dose of liothyronine is 10 mcg (or so) every 8 hours. Incidentally, an IV form of this drug will not be readily obtainable. Also, on page 15 the dosage for levothyroxine should read 400 to 500 mcg. The dosages as listed in the article would be lethal.

Also misleading are separate name listings of levothyroxine. Levothyroxine is the same as L-thyroxine, one trade name of which is Synthroid.

Brian Kaatz, Pharm.D.
Clinical Pharmacist
Sioux Falls, S.D.

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On behalf of the entire AMA family, I would like to congratulate the South Dakota State Medical Association on its 100th anniversary. Great changes have been made by your association in this time to keep pace with the dynamic needs of your membership while continuing to guarantee quality health care for the people of South Dakota.

In recounting the successful ventures of this society, the association-sponsored South Dakota Medical School Endowment Fund, must be regarded as a major achievement. This fund has enabled numerous students to attend medical school, who otherwise might not have been financially able to do so. Perhaps more important, however, was its part in convincing the state legislature of the sincerity of South Dakota physicians in their desire to have the medical school expanded to a four year institution. Redesignated as such in 1974, the University of South Dakota Medical School completed the cycle that now offers comprehensive medical training as well as superior health care to the people of South Dakota.

It is worth noting some of the more important accomplishments of recent years including the formation of SODAPAC, an organization whose impact was positively felt in our most recent election. In addition, all members of the South Dakota Medical Association must take pride in the fiscal responsibility shown by your leadership resulting in the debt free purchase of your new headquarters in 1974. Certainly, organized medicine has come a long way in South Dakota since May of 1883 when the Association's assets totaled \$9.17!

You have every reason to be proud of the history and achievements of your medical association in its first one hundred years. I urge you to continue in your enthusiasm so that the future may even overshadow the successes of your past.

Best wishes and continued success,
James H. Sammons, M.D.

I am writing this letter to extend to you and the South Dakota State Medical Association my sincere thanks in presenting me with the generous scholarship. I am currently a Junior student and will find the money very helpful in my continued education at the USD School of Medicine. Again, the money is heartily appreciated and I am honored in receiving this award.

Sincerely yours,
Jeff Murray

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WARNINGS:

Drug dependence: Empirin with Codeine can produce drug dependence of the morphine type and, therefore, has the potential for being abused. Psychic dependence, physical dependence, and tolerance may develop upon repeated administration of this drug and it should be prescribed and administered with the same degree of caution appropriate to the use of other oral, narcotic-containing medications. Like other narcotic-containing medications, the drug is subject to the Federal Controlled Substances Act.

Use in ambulatory patients: Empirin with Codeine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. The patient using this drug should be cautioned accordingly.

Interaction with other central nervous system (CNS) depressants: Patients receiving other narcotic analgesics, general anesthetics, phenothiazines, other tranquilizers, sedative-hypnotics, or other CNS depressants (including alcohol) concomitantly with Empirin with Codeine may exhibit an additive CNS depression. When such combined therapy is contemplated, the dose of one or both agents should be reduced.

Use in pregnancy: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. Therefore, Empirin with Codeine should not be used in pregnant women unless, in the judgment of the physician, the potential benefits outweigh the possible hazards.

PRECAUTIONS:

Head injury and increased intracranial pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a pre-existing increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.

Acute abdominal conditions: The administration of Empirin with Codeine or other narcotics may obscure the diagnosis or clinical course in patients with acute abdominal conditions.

Allergic: Precautions should be taken in administering salicylates to persons with known allergies: patients with nasal polyps are more likely to be hypersensitive to aspirin.

Special risk patients: Empirin with Codeine should be given with caution to certain patients such as the elderly or debilitated, and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture, peptic ulcer, or coagulation disorders.

ADVERSE REACTIONS: The most frequently observed adverse reactions to codeine include light-headedness, dizziness, sedation, nausea and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include euphoria, dysphoria, constipation, and pruritus.

The most frequently observed reactions to aspirin include headache, vertigo, ringing in the ears, mental confusion, drowsiness, sweating, thirst, nausea, and vomiting. Occasional patients experience gastric irritation and bleeding with aspirin. Some patients are unable to take salicylates without developing nausea and vomiting. Hypersensitivity may be manifested by a skin rash or even an anaphylactic reaction. With these exceptions, most of the side effects occur after repeated administration of large doses.

DOSE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient. It may occasionally be necessary to exceed the usual dosage recommended below in cases of more severe pain or in those patients who have become tolerant to the analgesic effect of narcotics. Empirin with Codeine is given orally. The usual adult dose for Empirin with Codeine No. 2 and No. 3 is one or two tablets every four hours as required. The usual adult dose for Empirin with Codeine No. 4 is one tablet every four hours as required.

DRUG INTERACTIONS: The CNS depressant effects of Empirin with Codeine may be additive with that of other CNS depressants. See WARNINGS.



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This Is Your Medical Association

Three Aberdeen physicians, **Drs. Bernard C. Gerber, Albin Janusz, and Joseph Primrose**, were among the first in the nation to successfully complete the voluntary recertification examination of the American Board of Surgery. Of the 20,000 physicians in the United States who are board certified in surgery, participation was limited to only 450 volunteers for this first, four-hour, written examination held in Atlanta, Ga. last fall.

* * * *

A Rapid City internist, **Dr. Steven Stock**, has been named Medical Director for River Park of the Black Hills. Dr. Stock, a native of Huron, received his medical degree in 1975 from the University of Colorado School of Medicine in Denver and completed his internship and residency at St. Luke's Hospital in Denver, in 1973. He came to Rapid City in 1980 after two years in the Army.

* * * *

Joel B. Huber, M.D., Redfield, has been elected chief of staff by the medical staff of Community Memorial Hosp. **Edward D'Souza, M.D.** was elected vice-chairman and **Sterling Berg, M.D.** was elected secretary.

* * * *

Robert Brown, M.D. has joined the medical-dental staff at St. Luke's Hospital. Dr. Brown, a family practitioner, is a native of Saskatoon, Saskatchewan. He received his medical education from the Univ. of Saskatchewan and interned at St. Paul's Hospital, also in Saskatoon. Dr. Brown comes to Aberdeen with his wife and 3 children from Oliver, British Columbia.

* * * *

R. W. Honke, M.D., Jr., Sioux Falls, will be joining the Wagner Community medical group this summer. Dr. Honke received his medical degree from Southern Illinois University in Springfield in 1975. He served his internship while in the Army at Brooks Army Medical Center in San Antonio, Texas. Since 1978, he has been in a family practice residency program in Sioux Falls. Dr. Honke and his wife Anita are both natives of the Wagner area. They have three children.

Jose Michieli, M.D., of Watertown, was appointed to the Planning on Development Disabilities by the Governor.

* * * *

Rapid City physician, **George E. Sanmartin, M.D.**, has been elected to a three-year term on the Board of Governors of the 11,000 member American College of Cardiology.

* * * *

Yankton physician, **Brooks Ranney, M.D.**, has been nominated president-elect of the American College of Obstetricians and Gynecologists. Dr. Ranney is professor of obstetrics and gynecology and director of the residency program at the USD School of Medicine. Since 1948, Dr. Ranney has been chairman of the OB/GYN Dept. at the Yankton Clinic and Sacred Heart Hospital, where he also served as chief of staff in 1963. He is also a consultant in gynecology to the State Human Services Center. Dr. Ranney is the author of more than 70 papers and five book chapters on gynecology and obstetrics. He received his medical degree from Northwestern Univ. Medical School in 1940 and served in Europe as a medical officer during World War II.

* * * *

William O. Rossing, M.D., Sioux Falls, has been notified by the American College of Physicians that he has been granted recertification in the specialty of internal medicine.

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THE SOUTH DAKOTA JOURNAL OF MEDICINE

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Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema, and bronchospastic reactivity to aspirin, iodides, or other non-steroidal anti-inflammatory agents. Anaphylactoid reactions have occurred in such patients.

Warnings: Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. *Motrin* should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If *Motrin* must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Chronic studies in rats and monkeys have shown mild renal toxicity characterized by papillary edema and necrosis. Renal papillary necrosis has rarely been shown in humans treated with *Motrin*.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue *Motrin* and the patient should have an ophthalmologic examination, including central visual fields and color vision testing. **Fluid retention and edema** have been associated with *Motrin*; use with caution in patients with a history of cardiac decompensation or hypertension. *Motrin* is excreted mainly by the kidneys. In patients with renal impairment, reduced dosage may be necessary. Prospective studies of *Motrin* safety in patients with chronic renal failure have not been done. *Motrin* can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy. Patients should report signs or symptoms of **gastrointestinal ulceration** or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema. To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged **corticosteroid therapy** should have therapy tapered slowly when *Motrin* is added. The antipyretic, anti-inflammatory activity of *Motrin* may mask inflammation and fever.

Drug interactions. *Aspirin*: used concomitantly may decrease *Motrin* blood levels.

Coumarin: bleeding has been reported in patients taking *Motrin* and coumarin.

Pregnancy and nursing mothers: *Motrin* should not be taken during pregnancy nor by nursing mothers.

Adverse Reactions

The most frequent type of adverse reaction occurring with *Motrin* is gastrointestinal, of which one or more occurred in 4% to 16% of the patients.

Incidence Greater Than 1% (but less than 3%)—Probable Causal Relationship

Gastrointestinal: Nausea* epigastric pain* heartburn* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence); **Central Nervous System:** Dizziness*, headache, nervousness; **Dermatologic:** Rash* (including maculopapular type), pruritus; **Special Senses:** Tinnitus; **Metabolic/Endocrine:** Decreased appetite; **Cardiovascular:** Edema, fluid retention (generally responds promptly to drug discontinuation; see PRECAUTIONS).

Incidence Less Than 1%—Probable Causal Relationship**

Gastrointestinal: Gastric or duodenal ulcer with bleeding and/or perforation, gastrointestinal hemorrhage, melena, gastritis, hepatitis, jaundice, abnormal liver function tests; **Central Nervous System:** Depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma; **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome, alopecia; **Special Senses:** Hearing loss, amblyopia (blurred and/or diminished vision, scotomata, and/or changes in color vision) (see PRECAUTIONS); **Hematologic:** Neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs' positive), thrombocytopenia with or without purpura, eosinophilia, decreases in hemoglobin and hematocrit; **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure, palpitations; **Allergic:** Syndrome of abdominal pain, fever, chills, nausea and vomiting, anaphylaxis, bronchospasm (see CONTRAINDICATIONS); **Renal:** Acute renal failure in patients with preexisting, significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria; **Miscellaneous:** Dry eyes and mouth, gingival ulcer, rhinitis.

Incidence Less Than 1%—Causal Relationship Unknown**

Gastrointestinal: Pancreatitis; **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri; **Dermatologic:** Toxic epidermal necrolysis, photoallergic skin reactions; **Special Senses:** Conjunctivitis, diplopia, optic neuritis; **Hematologic:** Bleeding episodes (e.g., epistaxis, menorrhagia); **Metabolic/Endocrine:** Gynecomastia, hypoglycemic reaction; **Cardiovascular:** Arrhythmia (sinus tachycardia, sinus bradycardia); **Allergic:** Serum sickness, lupus erythematosus syndrome, Henoch-Schönlein vasculitis; **Renal:** Renal papillary necrosis.

*Reactions occurring in 3% to 9% of patients treated with *Motrin*. (Those reactions occurring in less than 3% of the patients are unmarked.)

**Reactions are classified under "Probable Causal Relationship" (PCR) if there has been one positive rechallenge or if three or more cases occur which might be causally related. Reactions are classified under "Causal Relationship Unknown" if seven or more events have been reported but the criteria for PCR have not been met.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

Dosage and Administration: Do not exceed 2400 mg per day. If gastrointestinal complaints occur, administer with meals or milk.

Rheumatoid arthritis and osteoarthritis, including flares of chronic disease: Suggested dosage is 300, 400, or 600 mg t.i.d. or q.i.d. Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for relief of pain.

Caution: Federal law prohibits dispensing without prescription.

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Points To Ponder

"You will always find something in the last place you look"

"The chance of a piece of bread falling with the buttered side up is directly proportional to the cost of the carpet"

"No matter how long or how hard you shop for an item, after you've bought it, it will be on sale somewhere cheaper"

"No one's life, liberty, or property are safe while the legislature is in session"

"The other line always moves faster"

"In order to get a loan, you must first prove you don't need it"

"When a broken piece of office equipment is demonstrated for the repairman, it will work perfectly"

"There's never time to do it right, but there's always time to do it over"

"Nature always sides with the hidden flaw"

"The light at the end of the tunnel is the headlamp of an oncoming train"

"Celibacy is not hereditary"

"Never play leapfrog with a unicorn"

"A Smith and Wesson beats four aces"

"If everything seems to be going well, you obviously don't know what the hell is going on"

"If more than one person is responsible for a miscalculation, no one will be at fault"

"Never argue with a fool, people might not know the difference"

"Where you stand on an issue depends on where you sit"

"Friends come and go, but enemies accumulate"

"If you try to please everybody, nobody will like it"

"Interchangeable parts- won't!"

"Leakproof seals- will!"

"Self-starters- will not!"

"Never eat prunes when you are famished"

SDAFP Memorial Lecture

SDAFP Board of Directors, has sanctioned the "SDAFP Memorial Lecture", to be given at each of the chapter sponsored Black Hills Seminars.

This lecture is to be given by an active, affiliate or resident affiliate member of SDAFP on a topic of the speaker's preference. Suggested topics are available from the Education Committee, based upon the cyclic core of knowledge for family practice.

Member applications for the privilege of being selected for this lecture must be available to the state office by April 1 of each year for the Summer Seminar and October 15 for the Winter Seminar. The application will be a letter of intent to be selected and an outline, with references, of the proposed lecture, including the title. The speaker selected for each of these lectures will be handled by the SDAFP Education Committee through a review process.

This memorial lecture, dedicated to former SDAFP members now deceased, will carry the honorarium award of \$200. Your participation is invited.

SDAFP Thirty Years Old

The Charter date of our constituent (state) chapter was January 1, 1951. An organizational meeting had been held in Sioux Falls on June 4, 1950. Watch for a commemorative event during the Black Hills Summer Seminar. Happy Anniversary!

Plan Now To Attend

The AAFP's Annual Scientific Assembly, September 21-24, 1981, with special events for spouses. This event will be held in Las Vegas, Nevada.

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Future Meetings

August

Antibiotic Review—1981, Sheraton Washington Hotel, Washington, D. C., Aug. 10-11. Contact: Sandy McMillan, 67 Peachtree Park Dr., Suite 221-C, Atlanta, GA 30309.

Arrhythmias and Cardiac Ischemia: Diagnosis & Management, Hyatt Regency, Montreal, Canada, Aug. 14-16. 13 hrs. AAFP & AMA Category I credits. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

The Sixth Annual Convention of the American College of International Physicians, Holiday Inn, Chicago, Ill., Aug. 20-23. 12 hrs. AMA Category I Credit. Contact: Am. College of International Phys., 3030 Lake Ave., Fort Wayne, IN 46805. Phone: (219) 424-7414.

Cardiac Rehabilitation, Sheraton Anaheim, Anaheim, CA, Aug. 21-22. 13 hrs. AAFP & AMA Category I credits. Fee: \$245. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: (800) 525-6646.

Recent Advances in Diabetes Management, Glacier Park Lodge, East Glacier, Mont., Aug. 29. 7-8 hrs. Category I credit. Contact: Stanlee Dull, Exec. Dir., Am. Diabetes Assoc., Box 2411, Great Falls, MT 59403. Phone: (406) 761-0908.

October

EKG Interpretation and Arrhythmia Management, Carousel Inn, Cincinnati, OH, Oct. 2-3. 13 hrs. AAFP & AMA Category I credits. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: 800-525-8646.

Laboratory Design Workshop, Minneapolis, MN, Oct. 5-9. Fee: \$600. Contact: Norman V. Steere & Assoc., Inc., 140 Melbourne Ave., SE, Minneapolis, MN 55414. Phone: (612) 378-2711.

The Sixth Annual International Body Imaging Conference, Hyatt Regency Hotel, Maui, Hawaii, Oct. 10-18. 25 hrs. Category I credit. Fee: \$365. Contact: Conference Secretary, Sixth Annual Internat'l. Body Imaging Conf., West Park Hosp., Dept. of Radiology, 22141 Roscoe Blvd., Canoga Park, CA 91304.

Cardiac Rehabilitation, Sheraton, Philadelphia, PA, Oct. 16-17. 13 hrs. AAFP & AMA Category I credits. Contact: IMEC, Div. of Postgrad. & CME, 64 Inverness Dr., E., Englewood, CO 80112. Phone: 800-525-8646.

Complications in OB/GYN, Mayo Foundation Outreach Program, McKennan Hosp. Aud., Sioux Falls, SD, Oct. 23. 6 hrs. AAFP & AMA Category I credits. Contact: Ruth A. Muchow, Edu. Center, McKennan Hosp., 800 E. 21st St., Sioux Falls, SD. Phone: (605) 339-8000.

49th Annual Postgraduate Assembly, The Red Lion Inn, Omaha, Neb., Oct. 26-28. Contact: Lorraine E. Seibel, Exec. Sec., Omaha Mid-West Clinical Society, 7363 Pacific St., #210-A, Omaha, NE 68114. Phone: (402) 397-1443.

November

Cancer 1981/Cancer 2001—An International Colloquium, Shamrock Hilton Hotel, Houston, TX., Nov. 10-14. Contact: Dr. C. Stratton Hill Jr., Conf. Coord., Rm. 115, UT M.D. Anderson Hosp. and Tumor Instit., 6723 Bertner Ave., Houston, TX 77030. Phone: (713) 792-2222.

Grand Canyon International Conference on the Treatment of Addictive Behavior, South Rim of the Grand Canyon, Ariz., Nov. 17-21. Fee: \$185.00. Contact: Mary Evilsizer, Marketing and Promotions, U. of New Mex., Bureau of Conf. and Instit., 805 Yale, N.E., Albuquerque, NM 87131. Phone: (505) 277-2931.

Oncology, Mayo Foundation Outreach Program, McKennan Hosp. Aud., Sioux Falls, SD, Nov. 30. Contact: Ruth Muchow, Ed. Center Coord., McKennan Hosp., 800 E. 21st St., Sioux Falls, SD. Phone: (605) 339-8000.

February

Rheumatology Seminar IV, Captiva Island, Florida, Feb. 27-Mar. 6, 1982. 20 hrs. AMA Category I credit. Fee: \$250. Contact: Dept. of CME & Program Serv., Minnesota Med. Assoc., Suite 400, 2221 University Ave., S.E., Minneapolis, MN 55414. Phone: (612) 378-1875.

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Volume XXXIV August 1981 Number 8



Transactions Of The South Dakota

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Examine Me.

During the past several years, I have heard my name mentioned in movies, on television and radio talk shows, and even at Senate subcommittee sessions. And I have seen it repeatedly in newspapers, magazines, and yes, best-sellers. Lately, whenever I see or hear the phrases "overmedicated society," "overuse," "misuse," and "abuse," my name is one of the reference points. Sometimes even *the* reference point.

These current issues, involving patient compliance or dependency-proneness, should be given careful scrutiny, for they may impede my overall therapeutic usefulness. As you know, a problem almost always involves improper usage. When I am prescribed and taken correctly, I can produce the effective relief for which I am intended.

Amid all this controversy, I ask you to reflect on and re-examine my merits. Think back on the patients in your practice who have been helped through your clinical counseling and prudent prescriptions for me. Consider your patients with heart problems, G.I. problems, and interpersonal problems who, when their anxiety was severe, have been able to benefit from the medication choice you've made. Recall how often you've heard, as a result, "Doctor, I don't know what I would have done without your help."

You and I can feel proud of what we've done together to reduce excessive anxiety and thus help patients to cope more successfully.

If you examine and evaluate me in the light of your own experience, you'll come away with a confirmation of your knowledge that I *am* a safe and effective drug when prescribed judiciously and used wisely.

For a brief summary of product information on Valium (diazepam/Roche) ®, please see the following page. Valium is available as 2-mg, 5-mg and 10-mg scored tablets.

Valium® diazepam/Roche

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Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety. Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic. Symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis, stiff-man syndrome; convulsive disorders (not for sole therapy).

The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma, may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other anti-depressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

The clearance of Valium and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported, should these occur, discontinue drug. Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect.

Adults: Anxiety disorders, symptoms of anxiety: 2 to 10 mg b.i.d. to q.i.d., alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed, adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) **Children:** 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam/Roche) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500, Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10, Prescription Paks of 50, available in trays of 10.



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Each gram contains: Aerosporin® (Polymyxin B Sulfate) 5,000 units, bacitracin zinc 400 units, neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base); special white petrolatum qs; in tubes of 1 oz and 1/2 oz and 1/32 oz (approx.) foil packets.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as a failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of non-susceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section). Complete literature available on request from Professional Services Dept. PML.



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PAIN AND TENSION...

Double fault for weekend warriors

ACE THE ACHE
with

Equagesic[®] IV

(meprobamate and ethoheptazine citrate with aspirin) Wyeth

Twofold analgesic action teamed with time-proven efficacy against concurrent anxiety and tension in patients with musculoskeletal disease.*

EQUAGESIC—Abbreviated Summary

INDICATIONS: Based on a review of this drug by the National Academy of Sciences—National Research Council and other information, FDA has classified the indications as follows:

Possibly effective for the treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease or tension headache.

Final classification of the less-than-effective indications requires further investigation.

The effectiveness of Equagesic in long-term use, i.e., more than four months, has not been assessed by systematic clinical studies. The physician should periodically reassess usefulness of the drug for the individual patient.

CONTRAINDICATIONS: Equagesic should not be given to individuals with a history of sensitivity or severe intolerance to aspirin, meprobamate, or ethoheptazine citrate.

WARNINGS: Careful supervision of dose and amounts prescribed for patients is advised, especially with those patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g., alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on or habituation to the drug. Where excessive dosage has continued for weeks or months, dosage should be reduced gradually rather than abruptly stopped, since withdrawal of a "crutch" may precipitate withdrawal reaction of greater proportions than that for which the drug was originally prescribed. Abrupt discontinuance of doses in excess of the recommended dose has resulted in some cases in the occurrence of epileptiform seizures.

Special care should be taken to warn patients taking meprobamate that tolerance to alcohol may be lowered with resultant slowing of reaction time and impairment of judgment and coordination.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with the use

of minor tranquilizers (meprobamate, chlorthalidone, and diazepam) during the first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physicians about the desirability of discontinuing the drug.

Meprobamate passes the placental barrier. It is present both in umbilical-cord blood and in near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breast-feeding patients, the drug's higher concentration in breast milk as compared to maternal plasma levels should be considered.

Preparations containing aspirin should be kept out of the reach of children. Equagesic is not recommended for patients 12 years of age and under.

PRECAUTIONS: Should drowsiness, ataxia, or visual disturbance occur, the dose should be reduced. If symptoms continue, patients should not operate a motor vehicle or any dangerous machinery.

Suicidal attempts with meprobamate have resulted in coma, shock, vasomotor and respiratory collapse, and anuria. Very few suicidal attempts were fatal, although some patients ingested very large amounts of the drug (20 to 40 gm). These doses are much greater than recommended. The drug should be given cautiously, and in small amounts, to patients who have suicidal tendencies. In cases where excessive doses have been taken, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Hyperventilation has been reported occasionally. Any drug remaining in the stomach should be removed and symptomatic treatment given. Should respiration become very shallow and slow CNS stimulants, e.g., caffeine, Metrazol, or amphet-

amine, may be cautiously administered. If severe hypotension develops, pressor amines should be used parenterally to restore blood pressure to normal levels.

ADVERSE REACTIONS: A small percentage of patients may experience nausea with or without vomiting and epigastric distress. Dizziness occurs rarely when meprobamate and ethoheptazine citrate with aspirin is administered in recommended dosage. The meprobamate may cause drowsiness but, as a rule, this disappears as therapy is continued. Should drowsiness persist and be associated with ataxia, this symptom can usually be controlled by decreasing the dose, but occasionally it may be desirable to administer central stimulants such as amphetamine or mephentermine sulfate concomitantly to control drowsiness.

A clearly related side effect to the administration of meprobamate is the rare occurrence of allergic or idiosyncratic reactions. This response develops, as a rule, in patients who have had only 1-4 doses of meprobamate and have not had a previous contact with the drug. Previous history of allergy may or may not be related to the incidence of reactions.

Mild reactions are characterized by an itchy urticarial or erythematous, maculopapular rash which may be generalized or confined to the groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema and fever have also been reported.

More severe cases, observed only very rarely, may also have other allergic responses, including fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case), and hyperthermia. Treatment should be symptomatic such as administration of epinephrine, antihistamine, and possibly hydrocortisone. Meprobamate should be stopped, and institution of therapy should not be attempted.

Rare cases have been reported where patients receiving meprobamate suffered from aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia. In nearly every instance reported, other toxic agents known to have caused these conditions have been associated with meprobamate. A few cases of leukopenia during

continuous administration of meprobamate are reported, most of these returned to normal without discontinuation of the drug.

Impairment of accommodation and visual acuity has been reported rarely.

OVERDOSE: Two instances of accidental or intentional significant overdose with ethoheptazine citrate combined with aspirin have been reported. These were accompanied by symptoms of CNS depression, including drowsiness and light-headedness, with uneventful recovery. However, on the basis of pharmacological data, it may be anticipated that CNS stimulation could occur. Other anticipated symptoms would include nausea and vomiting. Appropriate therapy of signs and symptoms as they appear is the only recommendation possible at this time. Overdose with ethoheptazine combined with aspirin would probably produce the usual symptoms and signs of salicylate intoxication. Observation and treatment should include induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions.

DESCRIPTION: Each Equagesic tablet contains 150 mg meprobamate, 75 mg ethoheptazine citrate and 250 mg aspirin.

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*This drug has been evaluated as possibly effective for this indication.

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Down with pain

Step up to reliable relief

for mild to moderate pain

Wygesic[®]

(65 mg propoxyphene HCl and 650 mg acetaminophen) Wyeth

More than twice as much acetaminophen as the leading combination plus a full therapeutic dose of propoxyphene...all in a convenient, economical single tablet.

WYGESIC—Abbreviated Summary

INDICATION: For the relief of mild-to-moderate pain.

CONTRAINDICATION: Hypersensitivity to propoxyphene or to acetaminophen.

WARNINGS: CNS ADDITIVE EFFECTS AND OVERDOSE: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, or other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. Toxic effects and fatalities have occurred following overdoses of propoxyphene alone or in combination with other CNS depressants. Most of these patients had histories of emotional disturbances or suicidal ideation or attempts, as well as misuse of tranquilizers, alcohol, or other CNS-active drugs. Caution should be exercised in prescribing large amounts of propoxyphene for such patients (see **Management of Overdosage**).

DRUG DEPENDENCE: Propoxyphene can produce drug dependence characterized by psychic dependence and less frequently physical dependence and tolerance. It will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics. The abuse liability of propoxyphene is qualitatively similar to codeine, although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine.

USAGE IN AMBULATORY PATIENTS: Propoxyphene may impair the mental and/or physical abilities required for potentially hazardous tasks, e.g., driving a car or operating machinery. Patients should be cautioned accordingly.

USAGE IN PREGNANCY: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. **INSTANCES OF WITHDRAWAL SYMPTOMS IN THE NEONATE HAVE BEEN REPORTED FOLLOWING USAGE DURING PREGNANCY.** Therefore, propoxyphene should not be used in pregnant women unless, in the

judgement of the physician, the potential benefits outweigh the possible hazards.

USAGE IN CHILDREN: Propoxyphene is not recommended for children because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric group.

PRECAUTIONS: Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine. The CNS depressant effect of propoxyphene may be additive with other CNS depressants, including alcohol.

ADVERSE REACTIONS: The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting. These seem more prominent in ambulatory than in nonambulatory patients, some of these reactions may be alleviated if the patient lies down. Other adverse reactions include constipation, abdominal pain, skin rashes, light-headedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances. The chronic ingestion of propoxyphene in doses over 800 mg per day has caused toxic psychoses and convulsions. Cases of liver dysfunction have been reported.

DRUG INTERACTIONS: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, and other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended (see **Warnings**). Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine.

MANAGEMENT OF OVERDOSAGE: **SYMPTOMS:** The manifestations of serious overdosage with propoxyphene are similar to those of narcotic overdosage and include respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, cyanosis), extreme somnolence progressing to stupor or coma, pupillary constriction, and circulatory collapse. In addition to these characteristics, which are reversed by narcotic antago-

nists such as naloxone, there may be other effects. Overdoses of propoxyphene can cause delay of cardiac conduction as well as focal or generalized convulsions, a prominent feature in most cases of severe poisoning. Cardiac arrhythmias and pulmonary edema have occasionally been reported, and apnea, cardiac arrest, and death have occurred.

Symptoms of massive overdosage with acetaminophen may include nausea, vomiting, anorexia, and abdominal pain, beginning shortly after ingestion and lasting for 12 to 24 hours. However, early recognition may be difficult since early symptoms may be mild and nonspecific. Evidence of liver damage is usually delayed. After the initial symptoms, the patient may feel less ill; however, laboratory determinations are likely to show a rapid rise in liver enzymes and bilirubin. In case of serious hepatotoxicity (jaundice, coagulation defects, hypoglycemia, encephalopathy, coma, and death may follow. Renal failure due to tubular necrosis, and myocardiopathy, have also been reported.

Ingestion of 10 grams or more of acetaminophen may produce hepatotoxicity. A 13-gram dose has reportedly been fatal.

TREATMENT: Primary attention should be given to the reestablishment of adequate respiratory exchange through provision of a patent airway and institution of assisted or controlled ventilation. The narcotic antagonists, naloxone, nalorphine, and levallorphan, are specific antidotes against the respiratory depression produced by propoxyphene. An appropriate dose of one of these antagonists should be administered preferably I.V., simultaneously with efforts at respiratory resuscitation and the antagonist should be repeated as necessary until the patient's condition remains satisfactory. In addition to a narcotic antagonist, the patient may require careful titration with an anticonvulsant to control seizures. Analeptic drugs (e.g., caffeine or amphetamine) should not be used because of their tendency to precipitate convulsions.

Oxygen, IV fluids, vasopressors and other supportive measures should be used as indicated. Gastric lavage may be helpful. Activated charcoal can absorb a significant amount of ingested propoxyphene. Dialysis is of little value in poisoning by propoxyphene alone. Acetaminophen is rapidly absorbed, and efforts to remove the drug from the body should not be delayed. Copious gastric lavage and/or induction of emesis may be indicated. Activated charcoal is probably ineffective unless administered almost immediately after acetaminophen ingestion. Neither forced diuresis nor hemodialysis appears to be effective in removing acetaminophen. Since acetaminophen in overdose may have an antidiuretic effect and may produce renal damage, administration of fluids should be carefully monitored to avoid overload. It has been reported that mercaptamine (cysteine) or other thiol compounds may protect against liver damage if given soon after overdosage (8-10 hours). N-acetylcysteine is under investigation as a less toxic alternative to mercaptamine, which may cause anorexia, nausea, vomiting, and drowsiness. Appropriate literature should be consulted for further information (JAMA 237:2406-2407, 1977). Clinical and laboratory evidence of hepatotoxicity may be delayed up to one week. Acetaminophen plasma levels and half-life may be useful in assessing the likelihood of hepatotoxicity. Serial hepatic enzyme determinations are also recommended.

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Transactions Of The South Dakota State Medical Association One Hundreth Annual Meeting May 28, 29, 30, 31, 1981

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REPORT OF THE BUDGET AND AUDIT COMMITTEE

May 29, 1981

Ramada Inn, Sioux Falls, SD

The meeting was called to order at 8:30 a.m. by Winston Odland, M.D., president. Present for roll call were Drs. Odland, Joseph Hamm, Duane Reaney, Richard Gere, Howard Saylor, G. E. Tracy and Robert Johnson and Patty Butler.

The committee reviewed the CPA audit prepared by McGladrey Henderickson & Company. Mr. Johnson reviewed the audit for the information of the committee and Dr. Hamm made several explanatory statements. Dr. Tracy moved that the Budget and Audit Committee approve the audit as submitted. The motion was seconded and carried.

The meeting was adjourned at 9:00 a.m.

FIRST COUNCIL MEETING MINUTES

10:00 a.m.

Thursday, May 28, 1981

Ramada Inn

Sioux Falls, South Dakota

The meeting was called to order by Chairman Richard Gere. Those present for roll call were Doctors Winston Odland, Bruce Lushbough, Durward Lang, Joseph Hamm, Duane Reaney, Richard Gere, Howard Saylor, G. E. Tracy, G. Robert Bartron, A. A. Lampert, Jr., R. C. Jahraus, David Buchanan, W. O. Rossing, R. E. Gunnarson, Guy Tam, Larry Sittner, Frank Messner, Gordon Held, Robert Ferrell, M. George Thompson, James Wunder, James Larson, Michael Pekas, D. G. Ortmeier, J. A. Eckrich, Maynard Porter, Lowell Hyland, Charles Hollerman, T. H. Sattler and Mr. Steve Ellwing.

Dr. Odland moved to accept the minutes of the previous meeting as published and distributed. The motion was seconded and carried.

I. WESTERN PHYSICIANS PURCHASING ASSOCIATION. The Council reviewed correspondence from Dr. John Kahle, president of WPPA. **Dr. Messner moved that the State Medical Association execute judgment against Western Physicians Purchasing Association. The motion was seconded and carried.**

II. MEDICAL RECORDS BILL PASSED DURING 1981 LEGISLATIVE SESSION. The Council reviewed this legislation and discussed their concerns regarding the release of patient records to patients and the physician/patient relationship. **Dr. Saylor moved that the Commission on Legislation and Governmental Relations monitor this law and its affect on physician/patient relations in South Dakota for possible future legislative change. The motion was seconded and carried.**

III. AMA ALTERNATE DELEGATE. The Council received a letter from Dr. Russell Harris resigning as AMA alternate delegate. **Dr. Tracy moved that the Council defer action on this until the second Council meeting on Sunday, May 31. The motion was seconded and carried.**

IV. CORRESPONDENCE FROM SOUTH DAKOTA CHAPTER AMERICAN ACADEMY OF PEDIATRICS ON AUTOMOTIVE SAFETY. The Council received a request from the Academy on Pediatrics for a resolution from the House of Delegates calling attention to the automotive safety problem and a promise of cooperation in the control of the high mortality, and that the State Medical Association be prepared to give testimony to the LRC on this problem when such hearings are scheduled. **Dr. Saylor moved that the Council reiterate its previous policy acknowledging that automotive safety for children is a problem and recommending that an educational program be developed rather than a legislative program. The motion was seconded and carried.**

V. PROPOSAL FROM BLUE CROSS. The Council reviewed a proposed information sheet which can be displayed in physicians' offices notifying patients that diagnostic tests when ordered by their physician may be performed in the hospital outpatient department and paid for by Blue Cross. The Council noted that neither Blue Cross nor Blue Shield will pay for the same tests when performed in a physician's office. Following discussion **Dr. Tracy moved that the executive office notify Blue Cross that it is the Council's feeling the proposal from Blue Cross could be misleading, discriminatory and not in the best interest of the patient, inasmuch as reimbursement is not provided when services are rendered in a physician's office. The motion was seconded and carried.**

VI. PRESENTATION BY KENNETH HELENBOLT, M.D., MEDICAL CONSULTANT, NORTH DAKOTA BLUE SHIELD AND MR. STEVE PREZELLA, VICE PRESIDENT OF ADMINISTRATION FOR NORTH DAKOTA BLUE SHIELD CONCERNING ADMINISTRATION OF THE MEDICARE PROGRAM. Dr. Helenbolt and Council members discussed problems which have occurred in the administration of the Medicare program by North Dakota Blue Shield including: 1) staffing, 2) computer and programming breakdowns, 3) federal guidelines and regulations, 4) completion of claim forms and claims processing, 5) surgical second opinion program, 6) limitation of liability provisions, 7) processing time required by North Dakota, 8) misinformation and lack of information provided by North Dakota to physicians and claimants. This was for the Council's information. Mr. Johnson requested that North Dakota Blue Shield provide the executive office with a copy of the Medicare regulations pertaining to the limitation of liability provision for review by the Association's legal counsel.

VII. MEMBERSHIP IN NATIONAL FEDERATION OF INDEPENDENT BUSINESS. **Dr. Saylor moved that the State Medical Association join the National Federation of Independent Business. The motion was seconded and following further discussion failed.**

VIII. HONORARY MEMBERSHIP. **Dr. Gere moved that O. J. Mabee, M.D. be named an honorary member of the State Medical Association. The motion was seconded and carried. Dr. Lang moved that George Smith, M.D. be named an honorary member of the State Medical Association. The motion was seconded and carried.**

IX. LETTER FROM A. A. LAMPERT, M.D. The Council

received a letter from Dr. Lampert in appreciation of action taken at their previous meeting naming Dr. Lampert to honorary membership in the State Medical Association.

X. REPORT OF THE LIAISON COMMITTEE. LIAISON COMMITTEE MEETING

9:45 a.m.
April 21, 1981

Joe Foss Building, Room 216
Pierre, South Dakota

The meeting was called to order at 9:45 a.m. by John Elston, M.D., Chairman. Those present for roll call were: John Elston, M.D., Robert Ferrell, M.D., Gerald Loos, M.D., Durward Lang, M.D., Frank Messner, M.D., and George Thompson, D.O. Also in attendance were Richard Blair, Robert Hayes, M.D., Courtney Anderson, M.D., Charles Hollerman, M.D. and Robert Johnson.

Following roll call, a motion was made that the minutes of the previous meeting be accepted as printed and distributed.

The Committee then began discussion of the agenda items.

Item #1. Certificate of Need.

Dr. Elston asked that a brief report of the Certificate of Need program be presented for the Committee's information. Mr. Johnson reported to the Committee that the bill introduced into this legislative session was a compromise which had been arrived at after several hours of negotiation between the Medical Association, the Governor's office and the office of the Secretary of Health. It was pointed out that physicians' offices are specifically exempt from Certificate of Need program in South Dakota, and that such exemption has been a long-standing policy of the State Medical Association. The Committee specifically discussed the fact that a physician's office in Aberdeen may be purchasing a C.T. Scanner and was reassured by Secretary Blair that physicians' offices may purchase this particular piece of equipment or any other major medical equipment without obtaining a Certificate of Need.

Item #2. Mandatory Premarital Serology.

Dr. Lang reviewed for the Committee's information statistical data on premarital serologies which he obtained in the laboratory of Clinical Medicine. This information indicated that several positive findings had been reported to the Health Department and that the information previously presented to the Medical Association's Commission on Scientific Medicine and Legislative Commission appeared to be in conflict with the information which Dr. Lang had recently obtained. Mr. Corning, a representative of the Department of Health reported to the Committee that, in fact, positive findings had been reported to the Health Department, but that upon investigation, such positive reports reflected previously treated cases of syphilis and that none of the positive findings sent to the Health Department in recent years had involved new active cases of syphilis. After considerable discussion, the Committee recommended that the Commission on Scientific Medicine be presented with the updated data, both from the Laboratory of Clinical Medicine and the report from the Health Department, for their review and further recommendation to the Council.

Item #3: Review of Laboratory Service Legislation HB 1275.

The Committee then heard from Richard Melton of the State Health Lab concerning House Bill 1275, which had been introduced into the 1981 legislative session. The Committee expressed many grave concerns regarding certain sections of this proposed legislation and asked Dr. Melton where this bill had originated. Dr. Melton responded that the bill had been generated by interest within the Governor's staff and on the part of the Legislative Research Council staff, and that it had originally been intended to be only cleanup legislation. (It is to be noted that although changes were recommended, the bulk of the language in the proposal is the statute under which we are now operating). The Committee pointed out to Dr. Melton that certain sections of the bill certainly amounted to more than cleanup legislation and felt that an expression of the Committee's concern should be sent to the Interim Study Committee, which is now reviewing all state laboratory facilities.

Item #4. Reports of the Subcommittees.

Dr. Elston reported on the Subcommittee on Family Planning.

For the Committee's information, Dr. Elston reported that the Committee on Family Planning has had one prior meeting for purposes of orientation and that a subsequent meeting is scheduled in Aberdeen in approximately one week. The Committee has reviewed new literature to be passed out referring to GU diseases, and the Committee will discuss and advise on the appropriateness of this literature. Dr. Elston also informed the Committee that the family planning grant is up for a one-year renewal under Title 10. For purposes of general information, Dr. Elston pointed out that the county health nurses are doing most of the work in seeing patients and that doctors are contracted with to attend clinics only on an occasional basis. Dr. Elston also informed the Committee that former nurses of the MCH program in Rapid City aided by Western Health Systems had prepared a grant proposal enabling them to take over the Family Planning Program. Their proposal, however, was turned down by the Subarea Council and HSA.

Dr. Ferrell then reported on the Crippled Children's Subcommittee. Dr. Ferrell reported to the Liaison Committee that at a recent meeting of the Subcommittee on the Crippled Children's Program the Committee recommended elimination of certain Crippled Children's Clinics, which were not deemed to be cost effective in the minds of the subcommittee. He recommended that the patients seen at these clinics, which were eliminated, be referred to private physicians; and further recommended to the Crippled Children's Program that South Dakota physicians be used for referral whenever possible in the future. Dr. Ferrell then reported on the new provider agreement which has been worked out between his committee and the Department of Health. This new provider agreement will allow physicians to bill their usual and customary fee. The Health Department will reimburse under the Crippled Children's Program on a fee schedule. The physician, however, will be able to bill the patient for the difference between the Health Department reimbursement and his usual and customary fee.

A report was then received on the Maternal and Child Health Program. It was reported that at their last meeting Sioux Valley Hospital appeared and gave the Committee an indepth review of their program and an update on the activities of that hospital. No action was called for.

The Subcommittee on PKU has not yet met.

A report from the Subcommittee on Hypertension Screening was given by Dr. Lang. He informed the Committee that one meeting of this subcommittee has been held primarily for organizational purposes and that Dr. Monfore of Miller had been elected Chairman of the subcommittee. Dr. Lang informed the Liaison Committee that a more detailed report would be submitted to them following their next meeting.

Item #5. Discussion of the Role of the Department of Health.

The Committee reviewed standing orders presented by Dr. Eldon Bell for their information. Dr. Hayes then informed the Committee that individual county officers may establish standing orders for their own county health nurse which vary from the standard format sent out by the Department of Health. The modified standing orders can be instituted by individual county health officers to more accurately reflect the functions which they wish the county health nurse in their area perform.

Dr. Hayes then presented a paper on the role of the Health Department in matters affecting public health. A lengthy discussion occurred, at which time the Committee expressed their general support for the Health Department's involvement in matters relating to the traditional role of public health, i.e. water hygiene, solid waste disposal, air quality, sewage disposal, hazardous waste, food inspection and the like. The Committee expressed concern as to the amount of duplication on the part of regulators in the field of health and expressed their hope that a method could be devised which would eliminate the many duplicative inspections which physicians, their clinics, and hospitals must be subjected to. It was pointed out that many of the duplications come about because of federal agencies, but it was again reiterated that if anything under the new block grant system could be done to eliminate such duplication, it would be in the best interest of everyone to do so.

The greatest debate occurred in the portion of document from

the Department of Health which related to the Health Department's role in direct health services. Many individual preferences were expressed. It was the general consensus of the group that the Health Department's role in tuberculin skin testing and venereal disease seem to be appropriate roles for the department; however, it was pointed out quite clearly to the representatives of the Department of Health that other programs such as well child assessment immunization clinics, vision and hearing screening programs, blood pressure screening programs and others should as much as possible be provided by the private sector and not become services provided by the Department of Health. The Committee also felt it would be an appropriate role for the Health Department to be involved in nuisance control such as parasitic infestations.

The Committee expressed grave reservations about the WIC Nutrition Program. The Committee then expressed the Health Department's role in emergency medical services. The Committee felt that although the present state-wide EMS governing board has functioned well, local control by the medical community would have far greater advantages than trying to administer the entire program at a state level. The Committee felt that any change in the statutes governing the county health officers and the role of the Health Department in public health should be considered in detail by the Commission on Legislation of the South Dakota State Medical Association in conjunction with the Council, and that this Committee, along with the other appropriate Commissions referred to, cooperate and coordinate our activities with the Department of Health in drafting such legislation.

The Committee then moved on to item #6 on its agenda, which was an indepth review of health planning and its future, both locally and nationally. Mr. Ed DeAntoni of the staff of the Department of Health reported to the Committee that South Dakota's HSA, which operates on a fiscal year budget of August 1, 1981 to August 1, 1982, will receive at least a fifty percent cut in funding effective August 1, 1981 and will be zero funded on August 1, 1982. Mr. DeAntoni informed the Committee that the State Health Planning & Development Agency in South Dakota will continue to be funded during the next fiscal year at its present level; however, the State Health Planning & Development Agency is geared to be phased out of existence on July 1, 1983.

The Committee then referred to item #7 on its agenda. Teleconferencing and AHEC. Dr. Hollerman and Dr. Courtney Anderson presented for the Committee's information a new program being developed by the Medical School for teleconferencing. There are twenty such lines which will be available for continuing education programs and conferences throughout the state. This is a program which has been modeled after a Wisconsin teleconferencing network, and pilot programs will be put on in various communities throughout the state in the very near future.

Dr. Hollerman then reviewed the present status of the AHEC program in South Dakota. Dr. Hollerman indicated that the Rapid City AHEC will be terminated at the end of this contract year. The Dean indicated serious reservations on his part with the present operation of the AHEC program in our state. He indicated a hope to restructure the AHEC program in South Dakota since he presently feels too much of the money is being spent on administration and not enough resources are being committed to programs of value to the people intended to be served.

Dr. Ferrell then suggested to Secretary Blair that the subcommittees on maternal and child health, crippled children's and family planning be combined. He indicated if these advisory or subcommittees could not be combined, that they at least hold combined meetings in an attempt to recognize the tremendous amount of time being devoted by members of this Committee to attending meetings of these various subcommittees and the Liaison Committee. It was also suggested that the Liaison Committee meet on a quarterly basis in an attempt to keep on top of the many changes which are anticipated within the Department of Health.

There being no further business, the meeting was adjourned at 4:30 p.m.

Following review of this report Dr. Tracy moved that the Council accept the Liaison Committee's report and extend special commendation to the members for their work on behalf of the members

of the State Medical Association. The motion was seconded and carried.

XI. REPORT OF THE COMMISSION ON PROFESSIONAL LIABILITY.

COMMISSION ON PROFESSIONAL LIABILITY MINUTES

1:30 p.m. Ramada Inn
Thursday, May 14, 1981 Sioux Falls, South Dakota

The meeting was called to order by A. A. Lampert, Jr., M.D., acting chairman. Those present for roll call were Doctors Morris Radaek, Dale Berkebile, E. W. Sanderson, Frank Alvine, James Hovland, A. A. Lampert, and Richard Wake. It was moved, seconded and carried to approve the minutes of the previous meeting as printed and distributed.

The Commission reviewed information provided by the St. Paul Companies. This was accepted for information only.

The Commission reviewed information provided by American Health Systems and discussed the presentation made by American Health Systems at the January Commission meeting. **DR. RADACK MOVED THAT THE COMMISSION REVIEW CURRENT INFORMATION ON THE MINNESOTA MEDICAL INSURANCE EXCHANGE AND OTHER SIMILAR RECIPROCALS ON A CONTINUING BASIS. THE MOTION WAS SECONDED AND CARRIED.**

The Commission reviewed the Florida statute which states that if a defendant in a malpractice suit is found not guilty, the plaintiff is responsible for both his and the defendant's legal fees provided the plaintiff is not indigent. Presently, this law is being tested in the Florida courts, and the Commission on Legislation and Governmental Relations will be monitoring this situation.

Information on the Georgia Risk Management Program was reviewed. The St. Paul Companies, which has approved this program, allows physicians who attend to receive a professional liability insurance credit in the amount of 10% for the first year's attendance, 8% for the second year and 7% for the third year. The Commission was apprised that the AMA is becoming involved in risk management programs and directed the executive office to obtain information from the AMA on such programs available in surrounding states. They also requested that Mr. Johnson discuss this with representatives from the North Central Conference to see if it might be feasible for the five state conference to jointly sponsor a risk management program. This will be discussed further at the next Commission meeting.

A discussion was held concerning the parameters of this Commission. It was the feelings of the members that while the State Association Bylaws are quite broad, specific recommendations must be approved by the Council before being implemented. In view of this and because the Commission feels that physicians in South Dakota should have an advocate or appeals body with regard to renewal of professional liability insurance, **DR. SANDERSON MOVED THAT THE COMMISSION RECOMMEND TO THE COUNCIL THAT THIS COMMISSION BE AVAILABLE FOR CONSULTATION TO THE PHYSICIANS IN THE STATE AND TO THE MALPRACTICE CARRIERS REGARDING PHYSICIAN PROFESSIONAL LIABILITY COVERAGE OR INDIVIDUAL PROFESSIONAL LIABILITY PROBLEMS. THE MOTION WAS SECONDED AND CARRIED.** The Commission also recommended that this proposal be reviewed and approved by the Association's legal counsel before being implemented.

The Commission briefly discussed statistics available on malpractice suits in South Dakota and requested that Mr. Johnson provide information from the Insurance Commissioner's office on the audit of the St. Paul Companies for review at the next meeting.

The meeting adjourned at 4:30 p.m.
Dr. Lampert as acting chairman of this commission presented the report. **Dr. Buchanan moved that the Council accept the report and its recommendations. The motion was seconded and carried.**

X. REPORT ON THE MEDICAL SCHOOL. Dr. Hollerman gave a brief report on the Medical School, the 1981 graduating class, the faculty recognition days scheduled for September 4 and 5 in Rapid City, the use of teleconferencing, an update on the

search for Chairman of the Family Practice Department, AHEC and the Medical School's Admissions Committee. This was for the Council's information. Dr. Lang expressed appreciation to Dr. Hollerman for polling the physicians regarding the inclusion of students on the Admissions Committee, and Dr. Hollerman indicated that the replies received were about 50-50 and that he would make a final decision on Monday, June 1.

XI. LETTER FROM GOVERNOR JANKLOW. Dr. Gere read a letter from Governor Janklow commending Dr. Odland and Mr. Johnson for their efforts on behalf of medicine and the citizens in South Dakota with regard to health programs and needs for the state of South Dakota.

The meeting adjourned at 12:00 noon.

SECOND COUNCIL MEETING MINUTES

10:45 A.M. Ramada Inn
Sunday, May 31, 1981 Sioux Falls, South Dakota

The meeting was called to order by R. C. Gere, M.D., Chairman of the Council. Those present for roll call were Doctors Bruce Lushbough, Durward Lang, Joseph Hamm, G. E. Traey, Howard Saylor, Winston Odland, J. A. Eekrieh, A. A. Lampert, Jr., David Buchanan, R. G. Gere, Guy Tam, Larry Sittner, R. E. Gunnarson, Lowell Hyland, Dennis Johnson, Gordon Held, Frank Messner, Robert Ferrell, A. J. Barrett, N. R. Whitney, M. George Thompson, James Wunder and Eldon Bell.

Dr. Saylor moved to dispense with the reading of the minutes of the previous meeting inasmuch as they will be published and distributed. The motion was seconded and carried.

Dr. Gere introduced the new Councilors present, Dr. J. A. Eekrieh from the Aberdeen District and Drs. Lowell Hyland and Dennis Johnson from the Sioux Falls District. Dr. Johnson is an alternate councilor seated in the absence of Dr. Michael Pekas.

I. ELECTION OF CHAIRMAN OF THE COUNCIL. Dr. Traey nominated Dr. Richard Gere. **Dr. Lushbough moved that nominations cease and a unanimous ballot be cast for Dr. Gere as chairman of the Council. The motion was seconded and carried.**

II. PROPOSED COUNCIL AND COMMISSION MEETING DATES FOR 1981-82. The following proposed meeting dates were provided to the council for information:

Council: September 25, 26, 1981
November 21, 1981
April 16, 17, 1982
May 20-23, 1981 (Annual Meeting)

Commission:
August 29, 1981 Commission on Medical Service
Commission on Legislation
September 25, 1981 Commission on Scientific Medicine
Commission on Internal Affairs
January 9, 1982 Commission on Legislation
Budget and Audit Committee
Executive Commission
March 27, 1982 Commission on Internal Affairs
Commission on Scientific Medicine
Commission on Medical Service

III. ELECTION OF SECRETARY-TREASURER. Nominations were in order to complete the unexpired term of Dr. Joseph Hamm as secretary-treasurer inasmuch as Dr. Hamm was elected vice president. Dr. Lang nominated Dr. William O. Rossing. **Dr. Odland moved that nominations cease and a unanimous ballot be cast for Dr. Rossing as secretary-treasurer of the State Medical Association. The motion was seconded and carried.**

IV. AMA ALTERNATE DELEGATE. The Council was apprised that Dr. Russell Harris withdrew his resignation as AMA alternate delegate so no action by the Council was necessary.

V. RESOLUTION FROM THE SOUTH DAKOTA SOCIETY OF INTERNAL MEDICINE. The following resolution was submitted to the Council for consideration:

WHEREAS, the AMA Department of Negotiations has offered and conducted introductory negotiations seminars on a cosponsored basis with state, county and specialty medical societies since 1977 and,

WHEREAS, each intensive two-day seminar program provides an overview of the negotiations process; the procedure, tactics, and strategies associated with the process; exercises; and case problems designed to develop negotiating knowledge, skills, and abilities and,

WHEREAS, these programs are offered to give groups a unique opportunity to provide a tangible membership benefit by presenting a program for physicians on negotiating and coping with others—such as fellow physicians, patients, hospital management, insurers, government agencies, and health planners,

BE IT RESOLVED, that the Council of the South Dakota State Medical Association investigate and correspond with the AMA Department of Negotiations on the feasibility of cosponsoring such a seminar for SDSMA members.

Dr. Gunnarson moved to refer this resolution to the Commission on Internal Affairs, Communications and Liaison for study and deliberation and request that they report back to the Council at the next Council meeting. The motion was seconded and carried.

VI. REVIEW OF GRIEVANCE COMMISSION FUNCTIONS. Dr. Reaney discussed previous council action which referred a study of the Grievance Commission's functions to the Commission on Internal Affairs, Communications and Liaison. Following discussion **Dr. Buchanan moved that this study not be referred to the Commission on Internal Affairs, Communications and Liaison but rather be referred to an ad hoc committee consisting of the Grievance Commission members. The motion was seconded and carried.**

VII. STATEWIDE CANCER REGISTRY. The South Dakota Health Systems Agency provided background material on a proposed statewide cancer registry in South Dakota and requested the Council's endorsement of this proposal. **Dr. Lushbough moved that this proposal be referred to the Commission on Scientific Medicine for review and recommendation. The motion was seconded and carried.**

VIII. ARCHIVES AND HISTORY COMMITTEE. The Council discussed the need for a committee to gather and organize historical material pertaining to the State Medical Association for future reference. **Dr. Hamm moved that an ad hoc committee be appointed to begin collecting and organizing historical material, and that a bylaw amendment be submitted to the House of Delegates at the 1982 meeting making this a standing commission of the State Medical Association. The motion was seconded and carried. Dr. Gunnarson moved that Dr. C. J. McDonald be named chairman of this ad hoc committee. The motion was seconded and carried.**

The meeting adjourned at 11:00 a.m.

REMARKS BY ROBERT KELLY, M.D.

Immediately following adjournment of the Council meeting, a member of the AMA Board of Trustees, addressed members of the South Dakota State Medical Association and included a slide presentation. Dr. Kelly presented information on a survey of AMA members which showed what members perceive is the AMA's purpose, what the AMA can do as compared to specialty societies and what the members feel the AMA should do. He reviewed the Board of Trustees recommendations for an increase in AMA dues, a change in dues structure for various categories of AMA members, the discontinuance of several standing Councils and a study on alternatives for the AMA interim meetings. Dr. Kelly encouraged physicians to continue active participation in the AMA and requested their help in promoting membership so the AMA can continue to be the representative body of the nation's physicians.

MINUTES OF THE FIRST HOUSE OF DELEGATES MEETING

Friday,
May 29, 1981

Ramada Inn
Sioux Falls, S.D.

The meeting was called to order at 8:30 a.m. by Howard Saylor,

Jr., M.D., Speaker of the House of Delegates. The following physicians were present for roll call: Drs. Winston Odland, Bruce Lushbough, Durward Lang, Joseph Hamm, Duane Reaney, Howard Saylor, Gerald Tracy, Russell Harris, G. Robert Bartron, A. A. Lampert, Jr., R. C. Jahraus, David Buchanan, R. G. Gere, William O. Rossing, Richard Gunnarson, Guy Tam, Larry Sittner, Gordon Held, Frank Messner, N. R. Whitney, Robert L. Ferrell, A. J. Barrett, M. George Thompson, James Wunder, Eldon Bell, David Seaman, John Christopher, Karlis Zvejnieks, James Rud, James Larson, Richard Wake, Curtis Wait, John David, Robert Hohm, Louis Karlen, Chris Moller, Maynard Porter, John Ochsenner, Gail Benson, Robert Talley, Neil Elkjer, Daniel Kennelly, Gene Koob, Lynn Henrickson, Dennis Johnson, R. I. Porter, Jay Hubner, Tom Olson, O. Myron Jerde, Thomas Krafka, Charles Tesar, Robert Westaby, Charles Loos, Louis Hogrefe, James Collins, E. A. Johnson, and students Daryls Hofer and Dave Faragher.

Dr. Loos moved to dispense with the reading of the minutes of the last meeting inasmuch as they have been published in the SOUTH DAKOTA JOURNAL OF MEDICINE. The motion was seconded and carried.

Dr. Saylor read a letter from the mayor of the City of Milbank congratulating the South Dakota State Medical Association on its 100th annual meeting. The first meeting was held in Milbank in 1881.

Dr. Odland, president of the South Dakota State Medical Association presented the following awards: Community Service Award—J. A. Muggly, M.D., Madison; Distinguished Service Award—Saul Friefeld, M.D., Brookings; C. B. Alford Award—T. H. Sattler, M.D.; Past President's Award—Duane Reaney, M.D.; 50 Year Award—Myron C. Tank, M.D.

Mrs. Leni Johnson, Auxiliary AMA-ERF Chairman, presented a check to Charles Hollerman, M.D., Dean of the USD School of Medicine, representing contributions from South Dakota physicians, Auxiliary members, and District Auxiliaries during the year 1980.

Dr. Eldon Bell of Webster, presented the original Whetstone Valley District Medical Society charter to the South Dakota State Medical Association for safekeeping in the Association archives.

Dr. Saylor then announced the appointments to the Nominating Committee which had been made previously by Winston Odland, M.D., president. The appointments were as follows:

District #1—John Christopher, M.D.
District #2—G. E. Tracy, M.D.
District #3—Richard Wake, M.D.
District #4—John Davis, M.D.
District #5—David Buchanan, M.D.
District #6—Richard Gere, M.D., Chairman
District #7—William Rossing, M.D.
District #8—Frank Messner, M.D.
District #9—A. J. Barrett, M.D.
District #10—M. George Thompson, M.D.
District #11—James Collins, M.D.
District #12—E. A. Johnson, M.D.

Dr. Saylor then made the following appointments to the remaining reference committees.

Reference Committee on Credentials, Resolutions and Memorials, and Reports of Officers and Councilors.

David Seaman, M.D., Chairman
Chris Moller, M.D.
Thomas Krafka, M.D.

Reference Committee on Reports of Commissions on Medical Service, Legislation and Governmental Relations

Curtis Wait, M.D., Chairman
Gene Koob, M.D.
R. I. Porter, M.D.

Reference Committee on Reports of Commissions on Scientific Medicine, Internal Affairs, Communications and Liaison, and Professional Liability

Myron Jerde, M.D., Chairman
Neil Elkjer, M.D.
Jay Hubner, M.D.

Reference Committee on Reports of Special Committees and Miscellaneous Business

R. E. Gunnarson, M.D., Chairman
Louis Karlen, M.D.
Louis Hogrefe, M.D.

Dr. Gere moved to dispense with the reading of the reports of the president, president elect, vice president, secretary treasurer, delegate and alternate delegate to the AMA, executive secretary, speaker of the house, councilor at large, chairman of the council, and councilors inasmuch as they have been published in the handbook. The motion was seconded and carried.

Mr. Johnson then read Resolution #3, introduced by the 7th District Medical Society, which was not included in the handbook.

RESOLUTION #3

TO: House of Delegates
South Dakota State Medical Association
FROM: Seventh District Medical Society
SUBJECT: Supplemental Health Insurance for Medical Recipients

REFERRED TO:

WHEREAS, many South Dakota senior citizens have purchased supplemental health insurance to help defray costs not covered by Medicare, and;

WHEREAS, many of our senior citizen patients are disillusioned when these supplemental policies do not provide the coverage they had expected, and;

WHEREAS, it appears that some of these policies should more clearly state the benefits available;

THEREFORE, BE IT RESOLVED that the Commission on Legislation and Governmental Relations review, along with the Division of Insurance of the State of South Dakota and other appropriate authorities, the differing types of supplemental health insurance sold in South Dakota and make appropriate recommendations for change to the Council of the South Dakota State Medical Association for further follow-up and action as may be appropriate.

Dr. Saylor referred Resolution #3 to Reference Committee #4, Special Committees and Miscellaneous Business.

Dr. Saylor referred Resolution #1, concerning smoking and submitted by the Whetstone Valley District Medical Society to Reference Committee #4, Special Committees and Miscellaneous Business.

RESOLUTION #1

TO: House of Delegates
South Dakota State Medical Association
FROM: Whetstone Valley District Medical Society
SUBJECT: Smoking at Medical Associations functions
REFERRED TO:

WHEREAS, it is an established scientific fact that tobacco and other smoke is harmful to the health of those who use the drugs; and

WHEREAS, it has been shown that non-users exposed to the exhaled smoke from those who do consume the drugs primarily endanger the health of the non-user in certain environments; and

WHEREAS, the non-smoker is entitled to the benefits of clean unpolluted air in certain environments; and

WHEREAS, the SDSMA has actively served the people of South Dakota and will continue to emphasize the importance of setting a proper public and private example for the health of the citizens of the State of South Dakota,

THEREFORE, BE IT RESOLVED that the South Dakota State Medical Association appropriately publicly proclaims that smoking will be strictly prohibited in all official business and professional meetings of the South Dakota State Medical Association.

Resolution #2, concerning drug education in the USD School of Medicine, introduced by the Black Hills District Medical Society, was read by Mr. Johnson and referred to Reference Committee #4, Special Committees and Miscellaneous Business.

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RESOLUTION #2

TO: House of Delegates
South Dakota State Medical Association
FROM: Black Hills District Medical Society
SUBJECT: Alcohol and Drug Dependency Educational Program at USD School of Medicine
REFERRED TO:
WHEREAS, Alcohol and Drug Dependency involves approximately 10% of the general population in South Dakota, and;
WHEREAS, such dependency affects the lives of a far greater number of persons, and;
WHEREAS, such dependency is an increasing problem in the field of medicine, and;
WHEREAS, the USD School of Medicine has a family practice oriented mandate, and;
WHEREAS, the medical students presently receive little educational exposure to the aforementioned problem and its management.
THEREFORE, BE IT RESOLVED that the South Dakota State Medical Association recommend to the Dean of the USD School of Medicine that formal course material on the subject be introduced in the didactic curriculum, and;
BE IT FURTHER RESOLVED that this educational exposure then be extended as a requirement in the clinical phase of training utilizing the Alcohol and Drug Dependency treatment centers now in existence throughout the state of South Dakota.

Bylaw Revision #1, concerning the Grievance Committee, was read by Mr. Johnson and referred to Reference Committee #4, Special Committees and Miscellaneous Business.

BYLAW REVISION #1

TO: House of Delegates
South Dakota State Medical Association
FROM: The Council
SUBJECT: Grievance Commission

ARTICLE X Commissions

Section 4.

In addition to the foregoing commissions, there shall be an Executive Commission and a Grievance Commission, a Credentials Commission and a Professional Liability Commission.

b: Grievance Commission—The Grievance Commission shall consist of five ((Past-Presidents)) (((members))) of the Association (((appointed by the President))) who shall serve five years except that the first appointments shall be one, two, three, four, and five years respectively. The commission shall receive grievances, investigate the facts, attempt to mediate the dispute or refer such grievances to the proper authorities, and to educate the profession in the ethics of the practice of medicine. Rules of procedure shall be submitted to the Council for approval.

Dr. Saylor then referred the remaining reports in the handbook to the appropriate reference committee.

Several announcements were made by Dr. Saylor concerning the events to be held during the centennial annual meeting.

The meeting adjourned at 9:45 a.m.

MINUTES OF THE SECOND HOUSE OF DELEGATES MEETING

9:30 A.M. Ramada Inn
Sunday, May 31, 1981 Sioux Falls, South Dakota

The meeting was called to order by speaker of the House, Dr. Howard Saylor. Those present for roll call were Doctors Winston Odland, Bruce Lushbough, Durward Lang, Joseph Hamm, Howard Saylor, Gerald Tracy, Duane Reaney, A. A. Lampert, Jr., R. C. Jahraus, David Buchanan, Richard Gere, Richard Gunnarson, Guy Tam, Larry Sittner, Frank Messner, Nathaniel Whitney, Robert Ferrell, A. J. Barrett, M. George Thompson, James Wunder, Eldon Bell, David Seaman, John Christopher, Karlis Zvejnieks, James Rud, John Davis, Louis Karlen, E. A. Hofer,

Chris Moller, Maynard Porter, John Ochsner, Gail Benson, Robert C. Johnson, Robert Talley, James Oakland, Neil Elkjer, Daniel Kennelly, K. Gene Koob, Dennis Johnson, R. I. Porter, Jay Hubner, Tom Olson, O. M. Jerde, Thomas Krafka, Charles Tesar, Robert Westaby, George Wood, Louis Hogrefe, E. A. Johnson and Darlys Hofer, student delegate, and Dr. Rande Short, resident delegate.

Dr. Gere moved to dispense with the reading of the minutes of the last meeting inasmuch as they will be published and distributed. The motion was seconded and carried.

Dr. Saylor introduced Dr. Charles McCarthy, president of the Minnesota Medical Association, to the House. Dr. Saylor then introduced Dr. Robert Kelly, AMA Board of Trustees member from Minnesota, to the House. Dr. Kelly discussed his appointment to the Joint Commission on Accreditation of Hospitals' Accreditation Committee and the changes which he hopes to see implemented in the accreditation process. He reviewed changes which he foresees in HMO's, PSRO's, and HSA's and encouraged physicians to continue participation in health planning on a local level. He discussed other AMA activities and proposed changes in staffing and commissions, and encouraged all physicians to join the AMA and be active members so the AMA will truly represent practicing physicians at the national level.

Dr. Gere read the report of the Nominating Committee.

REPORT OF THE NOMINATING COMMITTEE

The Nominating Committee submits the following recommendations for the consideration of the House of Delegates:

COUNCILORS

Aberdeen District #1	J. A. Eckrich, Jr., M.D.
Brookings-Madison District #3	A. A. Lampert, Jr., M.D.
Huron District #5	David Buchanan, M.D.
Mitchell District #6	Richard Gere, M.D.
Sioux Falls District #7	Michael Pekas, M.D.
	Lowell Hyland, M.D.
Black Hills District #9	Roger Millea, M.D.

ALTERNATE COUNCILORS

Aberdeen District #1	Juan Chavier, M.D.
Brookings-Madison District #3	Curtis Wait, M.D.
Huron District #5	G. Robert Bell, M.D.
Sioux Falls District #7	Dennis Johnson, M.D.
Black Hills District #9	Ed James, M.D.

OFFICERS

President Elect	Durward Lang, M.D.
Vice President	Joseph Hamm, M.D.
Speaker of the House	Howard Saylor, M.D.

ANNUAL MEETING SITE

1982—Rapid City
1983—Sioux Falls
1984—Rapid City

The Nominating Committee recommends that the State Medical Association membership be surveyed in 1981 regarding desirable sites for future annual meetings.

Respectfully submitted,
NOMINATING COMMITTEE
R. G. Gere, M.D., Chairman
John Christopher, M.D.
G. E. Tracy, M.D.
Richard Wake, M.D.
John Davis, M.D.
David Buchanan, M.D.
W. O. Rossing, M.D.
Frank Messner, M.D.
A. J. Barrett, M.D.
George Thompson, D.O.
James Collins, M.D.
E. A. Johnson, M.D.

Dr. Larson moved to accept the report of the Nominating Committee. The motion was seconded and carried.



For the pain of osteoarthritis
the proven power of

Motrin[®]
ibuprofen, Upjohn
600 mg Tablets
One tablet t.i.d.

Please see the following page for a brief summary of prescribing information.

Upjohn

Motrin® Tablets (ibuprofen, Upjohn)

Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema, and bronchospastic reactivity to aspirin, iodides, or other non-steroidal anti-inflammatory agents. Anaphylactoid reactions have occurred in such patients.

Warnings: Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. *Motrin* should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If *Motrin* must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Chronic studies in rats and monkeys have shown mild renal toxicity characterized by papillary edema and necrosis. Renal papillary necrosis has rarely been shown in humans treated with *Motrin*.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue *Motrin* and the patient should have an ophthalmologic examination, including central visual fields and color vision testing. **Fluid retention and edema** have been associated with *Motrin*; use with caution in patients with a history of cardiac decompensation or hypertension. *Motrin* is excreted mainly by the kidneys. In patients with renal impairment, reduced dosage may be necessary. Prospective studies of *Motrin* safety in patients with chronic renal failure have not been done. *Motrin* can inhibit **platelet aggregation** and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy. Patients should report signs or symptoms of **gastrointestinal ulceration** or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema. To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged **corticosteroid therapy** should have therapy tapered slowly when *Motrin* is added. The anti-pyretic, anti-inflammatory activity of *Motrin* may mask inflammation and fever.

Drug interactions. *Aspirin*: used concomitantly may decrease *Motrin* blood levels.

Coumarin: bleeding has been reported in patients taking *Motrin* and coumarin.

Pregnancy and nursing mothers: *Motrin* should not be taken during pregnancy nor by nursing mothers.

Adverse Reactions

The most frequent type of adverse reaction occurring with *Motrin* is gastrointestinal, of which one or more occurred in 4% to 16% of the patients.

Incidence Greater Than 1% (but less than 3%)—Probable Causal Relationship

Gastrointestinal: Nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence); **Central Nervous System:** Dizziness,* headache, nervousness; **Dermatologic:** Rash* (including maculopapular type), pruritus; **Special Senses:** Tinnitus; **Metabolic/Endocrine:** Decreased appetite; **Cardiovascular:** Edema, fluid retention (generally responds promptly to drug discontinuation; see PRECAUTIONS).

Incidence Less Than 1%—Probable Causal Relationship**

Gastrointestinal: Gastric or duodenal ulcer with bleeding and/or perforation, gastrointestinal hemorrhage, melena, gastritis, hepatitis, jaundice, abnormal liver function tests; **Central Nervous System:** Depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma; **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome, alopecia; **Special Senses:** Hearing loss, amblyopia (blurred and/or diminished vision, scotomata, and/or changes in color vision) (see PRECAUTIONS); **Hematologic:** Neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs' positive), thrombocytopenia with or without purpura, eosinophilia, decreases in hemoglobin and hematocrit; **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure, palpitations; **Allergic:** Syndrome of abdominal pain, fever, chills, nausea and vomiting, anaphylaxis, bronchospasm (see CONTRAINDICATIONS); **Renal:** Acute renal failure in patients with preexisting, significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria; **Miscellaneous:** Dry eyes and mouth, gingival ulcer, rhinitis.

Incidence Less Than 1%—Causal Relationship Unknown**

Gastrointestinal: Pancreatitis; **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri; **Dermatologic:** Toxic epidermal necrolysis, photoallergic skin reactions; **Special Senses:** Conjunctivitis, diplopia, optic neuritis; **Hematologic:** Bleeding episodes (e.g., epistaxis, menorrhagia); **Metabolic/Endocrine:** Gynecomastia, hypoglycemic reaction; **Cardiovascular:** Arrhythmia (sinus tachycardia, sinus bradycardia); **Allergic:** Serum sickness, lupus erythematosus syndrome, Henoch-Schönlein vasculitis; **Renal:** Renal papillary necrosis.

*Reactions occurring in 3% to 9% of patients treated with *Motrin*. (Those reactions occurring in less than 3% of the patients are unmarked.)

**Reactions are classified under "Probable Causal Relationship" (PCR) if there has been one positive rechallenge or if three or more cases occur which might be causally related. Reactions are classified under "Causal Relationship Unknown" if seven or more events have been reported but the criteria for PCR have not been met.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

Dosage and Administration: Do not exceed 2400 mg per day. If gastrointestinal complaints occur, administer with meals or milk.

Rheumatoid arthritis and osteoarthritis, including flares of chronic disease: Suggested dosage is 300, 400, or 600 mg t.i.d. or q.i.d. Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for relief of pain.

Caution: Federal law prohibits dispensing without prescription.

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Dr. Seaman read the report of the Reference Committee on Credentials, Resolutions and Memorials and Reports of Officers and Councilors.

REPORT OF THE REFERENCE COMMITTEE ON CREDENTIALS, RESOLUTIONS AND MEMORIALS AND REPORTS OF OFFICERS AND COUNCILORS

The following delegates, alternates, officers and councilors of the South Dakota State Medical Association were present: Doctors Winston Odland, Bruce Lushbough, Durward Lang, Joseph Hamm, Duane Reaney, Howard Saylor, Gerald Tracy, Russell Harris, G. Robert Bartron, A. A. Lampert, Jr., R. C. Jahraus, David Buchanan, R. G. Gere, William O. Rossing, Richard Gunnarson, Guy Tam, Larry Sittner, Gordon Held, Frank Messner, N. R. Whitney, Robert L. Ferrell, A. J. Barrett, M. George Thompson, James Wunder, Eldon Bell, David Seaman, John Christopher, Karlis Zvejnieks, James Rud, James Larson, Richard Wake, Curtis Wait, John David, Robert Hohm, Louis Karlen, Chris Moller, Maynard Porter, John Ochsner, Gail Benson, Robert Talley, Neil Elkjer, Daniel Kennelly, Gene Koob, Lynn Henrikson, Dennis Johnson, R. I. Porter, Jay Hubner, Tom Olson, O. Myron Jerde, Thomas Krafa, Charles Tesar, Robert Westaby, Charles Loos, Louis Hogrefe, James Collins, E. A. Johnson, and students Daryls Hofer and Dave Faragher.

A quorum was present for the meeting of the House of Delegates. Total registration for the convention is 338, including 200 physicians, 11 guests, 119 Auxiliary members and 8 sponsors.

The committee submits the following resolution for the consideration of the House of Delegates:

WHEREAS, the Seventh District Medical Society, the Seventh District Auxiliary and the Yankton District Auxiliary members have been so thorough in making arrangements for the success of the combined meeting of our Centennial anniversary,

BE IT RESOLVED, that the South Dakota State Medical Association give its voice in appreciation and thanks to the local physicians in the Seventh District and the members of the Seventh District Auxiliary and the Yankton District Auxiliary.

WHEREAS, the management of the Ramada Inn has been so cooperative in providing facilities for the success of the Centennial annual meeting of the South Dakota State Medical Association,

BE IT RESOLVED, that the South Dakota State Medical Association extend its thanks and appreciation to the Ramada Inn.

WHEREAS, the Sioux Falls Tribune, the Sioux Falls Argus Leader, KELO TV, KSFY TV, KSOO radio, KKRC radio, KRSS radio, KELO radio, KXRB radio and KNWC radio have been most cooperative in presenting the public news of the Centennial annual meeting of the South Dakota State Medical Association.

BE IT RESOLVED, that the South Dakota State Medical Association extend its thanks to the Sioux Falls Tribune, the Sioux Falls Argus Leader, KELO TV, KSFY TV, KSOO radio, KKRC radio, KRSS radio, KELO radio, KXRB radio and KNWC radio.

WHEREAS, the Minnehaha Country Club has been most cooperative in providing facilities for the golf tournament and Thursday evening stag party and the Auxiliary luncheon on Saturday,

BE IT RESOLVED, that the South Dakota State Medical Association extend its thanks and appreciation to the Minnehaha Country Club.

BE IT RESOLVED, that \$50 be donated to the South Dakota Medical School Endowment Association in memory of the following physicians who died during the past year:

William Sercl, M.D.
M. L. Spain, M.D.
Claude Dulaney, M.D.
R. V. Avotins, M.D.
Fred Leigh, M.D.

The committee submits the following resolution for the consideration of the House of Delegates:

WHEREAS, the members of the State Medical Associations' Centennial Committee: Loren Amundson, M.D., A. J. Barrett M.D., Mrs. Marie Hovland and James Larson M.D. have graciously given their time and talent in preparation of our 100th anniversary,

BE IT RESOLVED, that the South Dakota State Medical Association give its voice in appreciation and thanks to these friends.

The Committee reviewed the reports of the officers and councilors and recommends they be accepted as submitted.

The Committee would also like to recognize the outstanding work and ability of our Executive Secretary, Robert Johnson and his staff during the last year in conducting the South Dakota State Medical Association.

Respectfully submitted,

REFERENCE COMMITTEE ON CREDENTIALS, RESOLUTIONS AND REPORTS OF OFFICERS AND COUNCILORS

David Seaman, M.D., Chairman

Chris Moller, M.D.

Thomas Krafa, M.D.

Dr. Moller moved to accept the report of the Reference Committee on Credentials, Resolutions and Memorials and Reports of Officers and Councilors. The motion was seconded and carried. Dr. Odland moved that the House commend the Centennial Committee for their excellent efforts on behalf of the annual meeting. The motion was seconded and carried.

Dr. Koob read the report of the Reference Committee on Reports of the Commission on Medical Service and the Commission on Legislation and Governmental Relations.

REPORT OF THE REFERENCE COMMITTEE ON REPORTS OF THE COMMISSION ON MEDICAL SERVICE AND THE COMMISSION ON LEGISLATION AND GOVERNMENTAL RELATIONS

The Reference Committee carefully reviewed the report of the Commission on Legislation and Governmental Relations. The Reference Committee recommends the acceptance of the report of the Commission on Legislation and Governmental Relations and would like to commend the Commission for their work during the year.

The Reference Committee reviewed the report of the Commission on Medical Service. The Reference Committee recommends the acceptance of the report of the Commission on Medical Service and would like to commend the Commission for their work during the year.

Respectfully submitted,

REFERENCE COMMITTEE ON REPORTS OF THE COMMISSION ON MEDICAL SERVICE AND THE COMMISSION ON LEGISLATION AND GOVERNMENTAL RELATIONS

Curtis Wait, M.D., Chairman

K. Gene Koob, M.D.

Richard Porter, M.D.

Dr. Ochsner moved to accept the report of the Reference Committee on Reports of the Commission on Medical Service and the Commission on Legislation and Governmental Relations.

Dr. Jerde read the report of the Reference Committee on Reports of the Commission on Scientific Medicine, the Commission on Internal Affairs, Communications and Liaison and the Commission on Professional Liability.

REPORT ON THE REFERENCE COMMITTEE ON REPORTS OF THE COMMISSIONS ON SCIENTIFIC MEDICINE, INTERNAL AFFAIRS, COMMUNICATIONS AND LIAISON AND PROFESSIONAL LIABILITY

The Reference Committee reviewed the report of the Commission on Scientific Medicine. The Reference Committee recommends acceptance of this report.

The Reference Committee reviewed the report of the Commission on Internal Affairs, Communications and Liaison. The

Reference Committee recommends the acceptance of this report.

The Reference Committee reviewed the report of the Commission on Professional Liability. The Reference Committee recommends acceptance of this report.

The Reference Committee wishes to commend the members of these three Commissions for their diligent efforts throughout the past year on behalf of the members of the State Association.

Respectfully submitted,

REFERENCE COMMITTEE ON REPORTS OF THE
COMMISSIONS ON SCIENTIFIC MEDICINE, INTER-
NAL AFFAIRS, COMMUNICATIONS AND LIAISON,
AND PROFESSIONAL LIABILITY

Myron Jerde, M.D., Chairman

Neil Elkjer, M.D.

Jay Hubner, M.D.

Dr. Tesar moved to accept the report of the Reference Committee on Reports of the Commission on Scientific Medicine, the Commission on Internal Affairs, Communications and Liaison and the Commission on Professional Liability.

Dr. Gunnarson read the report of the Reference Committee on Reports of Special Committees and Miscellaneous Business.

REPORT OF THE REFERENCE COMMITTEE ON REPORTS OF SPECIAL COMMITTEES AND MISCELLANEOUS BUSINESS

The Reference Committee has reviewed and recommends acceptance of reports from: The Grievance Commission, The Long Range Planning Committee, The South Dakota Political Action Committee, The Endowment Association, and The Committee for Continuing Medical Education.

The Committee recommends that the House of Delegates considers affirmative action for the Amended Resolution #1 as stated:

WHEREAS, it is an established scientific fact that tobacco and other smoke is harmful to the health of those who use the drugs; and

WHEREAS, it has been shown that non-users exposed to the exhaled smoke from those who do consume the drugs primarily endanger the health of the non-user in certain environments; and

WHEREAS, the non-smoker is entitled to the benefits of clean unpolluted air in certain environments; and

WHEREAS, the SDSMA has actively served the people of South Dakota and will continue to emphasize the importance of setting a proper public and private example for the health of the citizens of the State of South Dakota,

THEREFORE, BE IT RESOLVED that the South Dakota State Medical Association actively discourage smoking in all official business and professional meetings of the State Association.

The Reference Committee recommends that the House of Delegates considers affirmative action for Amended Resolution #2 as stated:

WHEREAS, Alcohol and Drug Dependency involves approximately 10% of the general population in South Dakota, and;

WHEREAS, such dependency affects the lives of a far greater number of persons, and;

WHEREAS, such dependency is an increasing problem in the field of medicine, and;

WHEREAS, the USD School of Medicine has a family practice oriented mandate, and;

WHEREAS, the medical students presently receive some educational exposure to the aforementioned problem and its management.

THEREFORE, BE IT RESOLVED that the South Dakota Medical Association commend the Dean of the USD School of Medicine for the formal course material on the subject presently existing in the didactic curriculum, and;

BE IT FURTHER RESOLVED that this educational exposure then be extended preferably as a requirement in the clinical phase of training to utilize the Alcohol and Drug Dependency treatment centers now in

existence throughout the State of South Dakota.

The Reference Committee recommends that the House of Delegates considers affirmative action for Resolution #3 as stated:

WHEREAS, many South Dakota senior citizens have purchased supplemental health insurance to help defray costs not covered by Medicare, and;

WHEREAS, many of our senior citizen patients are disillusioned when these supplemental policies do not provide the coverage they had expected, and;

WHEREAS, it appears that some of these policies should more clearly state the benefits available;

THEREFORE, BE IT RESOLVED that the Commission on Legislation and Governmental Relations review, along with the Division of Insurance of the State of South Dakota and other appropriate authorities, the differing types of supplemental health insurance sold in South Dakota and make appropriate recommendations for change to the Council of the South Dakota State Medical Association for further follow-up and action as may be appropriate.

The Reference Committee recommends that the House of Delegates considers Amended Bylaw Revision #1 for a do pass action:

ARTICLE X Commissions

Section 4.

In addition to the foregoing commissions, there shall be an Executive Commission and a Grievance Commission, a Credentials Commission and a Professional Liability Commission.

b: Grievance Commission—The Grievance Commission shall consist of five past Presidents of the Association who shall serve five years except that the first appointments shall serve one, two, three, four, and five years respectively. Any Grievance Commission vacancy which cannot be filled by a past President shall then be filled from a pool of Association members which have been selected by the current Association President.

The Commission shall receive grievances, investigate the facts, attempt to mediate the dispute or refer such grievances to the proper authorities, and to educate the profession in the ethics of the practice of medicine. Rules of procedure shall be admitted to the Council for approval.

Respectfully submitted,

REFERENCE COMMITTEE ON REPORTS OF SPECIAL COMMITTEES AND MISCELLANEOUS BUSINESS

R. E. Gunnarson, M.D., Chairman

L. W. Karlen, M.D.

L. Hogrefe, M.D.

Dr. Whitney moved to adopt Resolution #1 as amended by the Reference Committee. The motion was seconded and carried. Dr. Tesar moved to adopt Resolution #2 as amended by the Reference Committee. The motion was seconded and carried. Dr. Tam moved to adopt Resolution #3 as presented. The motion was seconded and carried. The Reference Committee proposed a change in Bylaw Revision #1 from the written report. The amendment would read as follows:

ARTICLE X Commissions

Section 4.

In addition to the foregoing commissions, there shall be an Executive Commission and a Grievance Commission, a Credentials Commission and a Professional Liability Commission.

b: Grievance Commission—The Grievance Commission shall consist of five past Presidents of the Association who shall serve five years except that the first appointments shall be one, two, three, four and five years respectively. The president of the South Dakota State Medical Association shall appoint a past president to fill any Grievance Commission vacancy which may occur.

The Commission shall receive grievances, investigate the facts, attempt to mediate the dispute or refer such grievances to the proper authorities, and to educate the profession in the ethics of the practice of medicine. Rules of procedures shall be admitted

to the Council for approval."

Dr. Buchanan moved to adopt Bylaw Revision #1 as amended by the Reference Committee. The motion was seconded and carried. Dr. Karlen moved to adopt the amended report of the Reference Committee on Reports of Special Committees and Miscellaneous Business. The motion was seconded and carried.

Dr. Saylor administered the Oath of Office to Bruce Lushbough, M.D., 1981-82 president. Dr. Lushbough briefly addressed the House, thanking the members for their support and requesting continued cooperation for a successful year ahead.

Dr. Saylor introduced the new officers, councilors and alternate councilors for the coming year to the House members. Dr. Lushbough commended Dr. Winston Odland for his dedication and service to the State Medical Association as president during the Centennial year.

The meeting adjourned at 10:30 a.m.

REPORT OF THE PRESIDENT

In the year since assuming the presidency of the South Dakota State Medical Association, I have been active in all matters of business conducted by the Association on behalf of its members. I have attended all of the Council meetings, meetings of the House of Delegates, Executive Committee meetings, with exception of the January meeting, and have traveled to Chicago for the meetings of the American Medical Association and again to San Francisco for the winter meeting of the American Medical Association.

The events of the year, I would say, add up to one of orderly business for the Association. Whereas we did not have major crises develop during the year, we have been busy working to achieve the objectives of South Dakota physicians and the citizens of South Dakota. In the legislative area, challenges to the medical profession were not forthcoming. The bills which were supported and introduced were passed, all of which were reasonable and not self-serving. Our relationship with State government, particularly the Governor and the legislature, was good. We continue to have philosophic variance with the activities of the State of South Dakota Department of Health in many of their programs which are involved with health care delivery. We have not turned back the pages of bureaucratic involvement in health affairs by the state government; however, there has not been a great increase in such activity. We have, through our Liaison Committee with the South Dakota Department of Health, developed good communications to inform the Department of Health of the objectives of free enterprise medicine in the state of South Dakota. We have also kept them well informed about our obligation to the delivering of quality health care to all citizens of South Dakota. I call your attention to the transcript of the South Dakota State Medical Association's Council at their winter meeting.

The South Dakota State Medical Association has continued to support the University of South Dakota School of Medicine. The Association has, by unanimous action of the Council, agreed not to become involved in the internal financial arrangements by the School of Medicine. Allocation of appropriated dollars are to be the province of the Medical School regarding their use. The Medical Association has continued to oppose the use of federal funds to develop health care delivery systems in South Dakota. Specific recommendations regarding this have been made with reference to AHEC. From the standpoint of the Association's President, I am concerned about the future of AHEC in spite of reassurances which have been given by the AHEC organization.

I see, as a matter for continued concern, the need for developing a due process for all activities of the Medical Association and also for all subdivisions of health care delivery. There are deficiencies, at the present time, which are difficult, particularly in the area of peer review. The outcome of the 1980 election brought forth a resurgence of conservative attitude regarding the scope and cost of government. These philosophies, which are building strength with each day, will have a predictable effect on the practice of medicine. We can look forward to decreasing amounts of money being expended by the federal government for health care programs in the state of South Dakota. With this will come a decrease in the amount of federal paychecks to physicians of this state. They must insist upon increasing amounts of dereg-

ulation of the practice of medicine along with the decreased paychecks. To insure this, our best efforts will be required. Deregulation is exactly what we have asked for, a divestment from the influence of federal and state government. Our obligation to patients and citizens of South Dakota has not changed.

I am pleased to have had the honor to be the President of the South Dakota State Medical Association. I hope the attitudes, philosophies and actions I have portrayed have been in keeping with the true wishes of the membership. While I realize that new faces and new ideas await in the wings of this theater of medical activity, I pledge my future participation on your behalf.

Without reservation, I think we have the finest Executive Secretary and staff for the South Dakota State Medical Association that we could have. I continue to be amazed at the amount of work which they do on our behalf, and the smoothness of the conduct of the business of our Association. I regard them all as trusted friends. With best wishes for continued success of our Medical Association.

Respectfully submitted,
Winston B. Odland, M.D.
President

The Reference Committee reviewed the report of the President and recommends it be accepted as submitted.

REPORT OF THE PRESIDENT-ELECT

As President-Elect of the South Dakota State Medical Association, I have attended the meetings of the House of Delegates, the Council and the Executive Commission during the past year.

I have also attended the AMA Leadership Conference and the AMA Meeting in July of 1980.

In this Centennial Year, I am mindful of the responsibilities of leadership in our organization. I will do my best to serve you as President in the coming year, mindful of the dedication of those who have served in this role in the previous 100 years.

Respectfully submitted,
Bruce Lushbough, M.D.
President-Elect

The Reference Committee reviewed the report of the President-Elect and recommends it be accepted as submitted.

REPORT OF THE VICE PRESIDENT

On the occasion of our Centennial Year, I offer congratulations to our fellow members of the South Dakota State Medical Association. Each of us is fortunate and proud to be a part of a record of 100 years of service to the citizens of the territory and the State of South Dakota.

We will continue our opposition to interference and intervention by well-intentioned third parties who would insert themselves between the physician and the patient, and thereby diminish us both.

We begin our second Hundred Years on a hopeful note. Our elected leadership has perhaps perceived that citizens will no longer be enchanted by untried "solutions" from Washington accompanied by regulations designed chiefly for the benefit of the regulators.

We can be proud of the fact that we now find ourselves in the vanguard of the movement to return to more sensible solutions to our problems.

Congratulations to you all!

Respectfully submitted,
Durward M. Lang, M.D.
Vice President

The Reference Committee reviewed the report of the Vice President and recommends it be accepted as submitted.

REPORT OF THE SECRETARY-TREASURER

The Secretary-Treasurer has attended quarterly meetings of the Council and meetings of the Executive Commission, with the exception of those held in November of 1980.

Throughout the past year, the Secretary-Treasurer and the Executive Secretary have conferred on matters relating to the finances and fiscal planning for the Association. Timely infor-

mation has been transmitted to the Council for consideration.

Along with the Executive Secretary and his staff, the annual budget has been prepared and submitted to the Executive Commission for subsequent review by the budget committee and finally by the House of Delegates. The Secretary-Treasurer represented the Association on the advisory committee to the office of the Commissioner for Higher Education for the administration of the Health Professions Loan Fund of the Board of Regents. In that capacity, he participated in the telephone conferences of that group during the past year. The House of Delegates is advised that the total funding made available to students in all the health professions through loans by the 1980-81 legislature is \$300,000.

Significant improvement in the fiscal status of the South Dakota State Medical Association during the past year is the result of increased membership and careful attention to the expenses and investments of the Association. The membership is to be complimented for its efforts in recruiting. To a large measure, the financial success of the Association is the result of the commendable work of the Executive Secretary and the office staff. The net result is that a dues increase can be deferred for at least another year.

Respectfully submitted,
Joseph N. Hamm, M.D.
Secretary-Treasurer

The Reference Committee reviewed the report of the Secretary-Treasurer and recommended it be accepted as submitted.

REPORT OF THE CHAIRMAN OF THE COUNCIL

It is always difficult to pinpoint just what activities and business are done by the Council. Suffice it to say that they are many and varied. Due to the fact that membership in the South Dakota Medical Association is increasing, there seems to be more to do. However, with the increase also comes an increase in the number of councilors to help in the decisions. The districts are to be commended for sending very able physicians for their representatives.

The Council is also very fortunate to have very hard-working Commissions handling problems and making recommendations as to their disposal. The Commission chairmen and members are to be commended.

I would like to make note of the untimely death of Fred Leigh, M.D., a long-time member of the Council and a past President of our organization. It is physicians like Fred who put many hours in the Council that are necessary for continued growth of the South Dakota State Medical Association.

As usual, the Council cannot say enough kind things about our Executive Secretary, Mr. Robert Johnson, and his very able staff. Without these people, our job would require much more time and be a great deal more tedious.

Respectfully submitted,
Richard G. Gere, M.D.
Chairman of the Council

The Reference Committee reviewed the report of the Chairman of the Council and recommended it be accepted as submitted.

REPORT OF THE AMA DELEGATE

The South Dakota delegation to the AMA attended the Annual Meeting in Chicago in July 1980 and the Interim Meeting in San Francisco in December 1980. The Medical Association was represented by W. R. Taylor, M.D., Delegate; Gerald Tracy, M.D., Alternate Delegate; Winston B. Odland, M.D., President of the State Medical Association, and Robert D. Johnson, Executive Secretary. A report of each meeting has previously been sent to all members of the State Medical Association.

My term of office as Delegate ended December 31, 1980, and the Association will be represented by Gerald Tracy, M.D., Delegate, and Russell Harris, M.D., Alternate Delegate, in the future.

It has certainly been a pleasure and an honor to represent the South Dakota State Medical Association in recent years, and I particularly wish to thank and commend our Executive Secretary, Robert D. Johnson, for his unfailing effort and great ability. His assistance has been invaluable, and the South Dakota State

Medical Association is greatly blessed by his continuing efforts as our Executive Secretary.

Respectfully submitted,
W. R. Taylor, M.D.
AMA Delegate

The Reference Committee reviewed the report of the AMA Delegate and recommended it be accepted as submitted.

REPORT OF THE ALTERNATE AMA DELEGATE

As the Alternate Delegate to the national AMA from the South Dakota State Medical Association, I have attended the two national meetings, all of the Council meetings, and executive meetings during the past year. It has been my pleasure to sit in at the House of Delegates for our Delegate, Dr. Bill Taylor, and to participate in the ongoing affairs of the national AMA.

I am continually impressed with the importance of the American Medical Association to each individual practicing physician and the viable input from a small state such as South Dakota. It has been a privilege to serve in this capacity the last year.

Respectfully submitted,
Gerald E. Tracy, M.D.
Alternate Delegate

The Reference Committee reviewed the report of the Alternate AMA Delegate and recommended it be accepted as submitted.

REPORT OF THE SPEAKER OF THE HOUSE

At the annual meeting of the South Dakota State Medical Association in Aberdeen in 1980, I was elected Speaker of the House.

I have attended all of the meetings of the Council and the Executive Commission. I am looking forward to presiding at the Centennial Meeting in Sioux Falls and will make every effort to help keep this your Medical Association.

Respectfully submitted,
Howard L. Saylor, Jr., M.D.
Speaker of the House

The Reference Committee reviewed the report of the Speaker of the House and recommended it be accepted as submitted.

REPORT OF THE COUNCILOR AT LARGE

As immediate past President, the position of Councilor At Large has been most enjoyable, being able to attend Council meetings, and observe the machinations of the work of the South Dakota State Medical Association without the burden of executive responsibility.

I attended all four of the quarterly Council meetings during the year and shared in the Council's deliberations. I also attended all meetings of the Executive Committee of the Association.

Respectfully submitted,
Duane B. Reaney, M.D.
Councilor At Large

The Reference Committee reviewed the report of the Councilor at Large and recommended it be accepted as submitted.

REPORT OF THE EXECUTIVE COMMISSION

The Executive Commission of the South Dakota State Medical Association was convened on three occasions during 1981, the minutes of which are published in the transactions of the Association.

The first meeting of the South Dakota State Medical Association's Executive Committee took place on June 20 and 21, 1980 at Peters' Sunset Beach, Glenwood, Minnesota. The context of the meeting was basically that of planning and becoming acquainted, with the officers learning of their objectives for the Medical Association. Early budget estimations, planning for the centennial year, and general discussions of legislative priorities took place.

The next meeting of the Executive Commission was held on November 21 and 22, 1980 in Miller, South Dakota in conjunction with the Board of Directors of the South Dakota Hospital Association. The context of this meeting was a mutual discussion of legislative views and frank discussions about all current events,

which were of mutual concern to our organizations. The meeting was considered a great success with closer ties and communications developing between the Hospital Association and the Medical Association.

The final meeting of the Executive Commission took place in Sioux Falls on January 10, 1981, at which time the proposed budget was reviewed and expenditures recommended for the ensuing year. Legislative priorities were again discussed and agreed upon.

During the past year, it has been the policy of the Executive Commission to embrace all the items of emergency or budgetary significance. General discussions of planning and legislative priorities were considered. All matters of business conducted by the Executive Commission were presented to the Council and approved. It is to be noted that two additional members of the Executive Commission, the Delegate to the AMA and Alternate Delegate, were admitted by action of the Council and House of Delegates. As President of the South Dakota State Medical Association, I advocate the continued use of the Executive Commission to handle all the business of an emergency nature, budgetary affairs and very general business, which is enhancing the unification of the Medical Association.

Respectfully submitted,
Winston B. Odland, M.D.
Chairman
Executive Commission

The Reference Committee reviewed the report of the Executive Commission and recommended the acceptance of this report.

REPORT OF THE EXECUTIVE SECRETARY

During 1980-81, I had the privilege of visiting, along with Dr. Odland, most of our District Medical Societies. Again, I want to extend our thanks and sincere appreciation for the gracious hospitality and fine attendance shown us during our visits. I am continually encouraged by the support given by the districts, through involvement in community activities and concerns, which not only elevates the public's awareness of the local medical profession, but also lends credence to your State Medical Association as an effective leader in our state. On numerous occasions this past year, the staff at the executive office has called upon not only the officers and councilors, but also the district officers for assistance and advice. This assistance has been most appreciated in helping to establish the policies and directions which your State Association ultimately has taken. It is through this unity that the goals of medicine can most effectively be achieved and maintained.

All commissions of the State Medical Association met at least twice this past year, and a report of their activities is included in this Delegate's Handbook. As in previous years, a multitude of issues and concerns from various sources, in both the public and private sectors, were presented to the commissions. Without hesitation, I believe we should all commend these individuals for the essential service which they have provided to the medical profession. They have continued, without interruption, to perform an outstanding job in dealing with problems presented to the Medical Association. In addition, the Liaison Committee, the Budget and Audit Committee and the Long Range Planning Committee each have carried out their responsibilities in a most meritorious fashion. The chairmen of these commissions and committees should be commended for giving special consideration to the many problems with which they must deal. Through excellent attendance and participation by the individual members, the Association is able to establish policies and programs which best serve the medical community and the general public.

As in the past, a great deal of time and effort by the executive office has revolved around state and national legislative activities. The 1981 legislative session found in excess of fifteen bills directly affecting the medical profession in the state of South Dakota and the patients you serve. Because of our demonstrated concern and ability to help direct change in a meaningful way, the legislature continues to turn to the medical profession for more and greater leadership. This was evident in the support by the legislature for those pieces of legislation which we sponsored or opposed. A special note of thanks should be given to Dr. Steve Haas, Chair-

man of the Commission on Legislation and Governmental Relations, and to all members of the commission for their diligent work and outstanding efforts on behalf of the physicians throughout the state of South Dakota. As you are aware, the medical school went through a very thorough zero-based budgeting process this past year. In addition, there were several pieces of legislation introduced affecting the medical school including residency programs, the Department of Family Practice and the Medical Profession's Scholarship Program. With the tight financial situation which the state presently faces, many legislators are questioning the need for a four-year degree granting school of medicine. Without your continued support, it is my firm belief that the medical school may be unable to obtain the necessary funding to maintain the high quality program which was established by the legislature in 1974. On the national scene, the Association responded to requests from the AMA concerning amendments to the Health Planning Law, PL 96-79; specifically the amendment that extended certificate of need coverage to physicians' offices. In addition, contact was made with South Dakota's congressional delegation concerning national health insurance, cost containment and a wide array of other health related topics.

"1881-1981, One Hundred Years of Service to Dakota Territory" symbolizes the proud heritage of the South Dakota State Medical Association. Our history and record of success can be traced directly to our pioneer heritage. Although few of these lonely and isolated physicians could have foreseen the complexity of modern day medicine and the impact the government and the public would ultimately have upon their profession; they did, however, foresee the need for an organized professional association which could grow and expand to meet the needs of medicine, the public and the physicians of South Dakota. This was the goal that drew these rugged individualists together. This remains the purpose and objective of your State Medical Association. With its unique history of dedication, to both its members and the public, the Association is proud of the role it has played in advancing medicine. It is proud of its history, and even more proud

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of its promising future. Although the purpose of the Association has remained constant throughout ten decades, medicine's environment has changed radically. If we are to survive as a profession, we must continue our leadership in defining the direction of change. The health planning system, as we know it today, will most likely be replaced by a competitive health care delivery model. It is expected that efforts will be directed at training physicians in specialties that are in short supply; that Public Health Service hospitals will be closed; that "a cap" on yearly federal expenditures under the medicaid program will be imposed; and on and on. The future holds many exciting moments for all physicians; but, once again, a united stand on the part of medicine will undoubtedly assist the leadership of this profession in obtaining maximum results for the private practicing physicians. I would certainly be remiss if I did not extend a special thanks to the Centennial Committee, Doctors L. H. Amundson, A. J. Barrett, J. C. Larson and Mrs. Marie Hovland. They have generously given their time and effort in developing our centennial celebration.

As we commence our second century of existence, we must now re-evaluate the role of your State Medical Association. This past year has presented an increasing number of challenges to the medical profession, but it has also been a year of progress. There have been many accomplishments achieved which the profession can view with considerable pride. Your executive staff is devoted to the concept of providing as many services as possible to the individual physician, the public and the state. Your organization is, and will continue to be, beneficial to all South Dakotans. The officers and councilors of your Association are most deserving of special recognition for the time, effort and leadership they have provided for this Association during the past year.

Finally, to Dr. Odland, your President and my personal friend, I would like to extend my congratulations on a job well done. Your strong convictions have been a credit to the profession and the office of President. Your guidance and foresight have been invaluable.

Respectfully submitted,
Robert D. Johnson
Executive Secretary

The Reference Committee reviewed the report of the Executive Secretary. The Committee would like to recognize the outstanding work and ability of our Executive Secretary, Robert Johnson, and his staff during the last year in conducting the South Dakota State Medical Association.

REPORT OF THE FIRST DISTRICT COUNCILOR

The First District Medical Society held its first meeting of the year on September 3, 1980. Social hour and dinner with wives attending was enjoyed. Dr. Myron Walzak, Chief of Urology at Creighton University, presented a discussion on the evaluation of patients with hematuria. Dr. Joe Huber, a new family physician at Redfield, was introduced.

The October meeting was again held at Helen's California Kitchen with a dinner and formal meeting following. Considerable discussion was held concerning advertising in the Yellow Pages of the phone book, and the Board of Censors have been asked to review standards for advertising in the Yellow Pages.

The November meeting was held November 5. Our State President, Dr. Winston Odland, and Mr. Bob Johnson, Executive Secretary, were present for their official annual visit.

The December meeting consisted mainly of business and election of officers. Dr. Barry Welge was elected as President, Dr. Carlton Kom as Vice President, and Dr. Jay Bachmayer as Secretary-Treasurer. Three delegates elected were Doctors David Seaman, Granville Steele and John Christopher. Alternate delegates elected are Doctors Harvey Hart, Charles Pelton and Karlis Zvejnieks.

The February meeting was held on February 4, and Dr. Larry Edwards, Director of Infectious Diseases at the Rockford School of Medicine in Rockford, Illinois, presented a paper on treatment of infectious diseases.

The March meeting was held on March 4. Dr. Charles Holterman, Dean of the South Dakota School of Medicine, spoke on the current status of the Medical School and also discussed AHEC. Also, Mr. Donald Brekke, Program Director of the Area Health Education Centers, presented a more detailed talk on the possibility of the formation of an AHEC in Aberdeen. The Aberdeen District unanimously passed a resolution supporting the Certificate of Need application for St. Luke's Hospital for a C.T. scanner. The Secretary was directed to write a letter to the State Health Planning Board supporting this application by St. Luke's Hospital.

The April meeting was held on April 1, 1981. Dr. P. Steven Johnson and Dr. Robert Marschke of Sioux Falls presented discussions on breast cancer and lung cancer.

The meeting for May is planned to have a discussion of infectious diseases.

Respectfully submitted
B. C. Gerber, M.D.
Councilor, First District

The Reference Committee reviewed the report of the Councilor from the First District Medical Society and recommended it be accepted as submitted.

REPORT OF THE SECOND DISTRICT COUNCILOR

The Watertown District Medical Society does not meet in the months of June, July and August following the Annual State Meeting in 1980.

They met in September with a social meeting with wives in attendance.

The October meeting included a presentation by Dr. Howard Saylor from Huron, South Dakota on categorization of emergency medical services. That meeting also included an update on the Medical School by G. Robert Bartron, M.D.

The November 4, 1980 meeting was the official meeting with the visit of the South Dakota State Medical Association president, Winston Odland, M.D., Vice President, Durward Lang, M.D., and Executive Secretary, Bob Johnson, who gave a very complete and thorough update on affairs of the State Medical Association, particularly regarding governmental intervention into the practice of medicine.

The December meeting included a presentation by Dr. Paul Carpenter on "Aortic Insufficiency, Acute and Chronic." There was also election of officers for the year 1981. The officers elected were: President, Dr. M. C. Thompson; Vice President, Dr. Paul Larson; Secretary-Treasurer, Dr. Gerald Tracy; Delegate for two years, Dr. James Rud; Alternate Delegate for Dr. Rud, Dr. Bernie Hanson; Censor for two years, Dr. David Oey.

At the January meeting, in addition to regular business, Dr. Solberg gave a presentation on "Xenon Ventilation and Profusion Imaging for Ventilation Studies."

The February meeting, in addition to regular business, included a presentation by Joyce Sugrue, Public Health Nurse, who spoke about the public health nursing programs and held a lengthy question and answer period.

The March meeting included a presentation by Dr. Sam Assam, a neurosurgeon from Sioux Falls, who presented an interesting program on "Occult Lumbar Disc Disease."

The April meeting is expected to also have a scientific program.

The attendance at the Watertown District Medical Society remains extremely high, and the society is continually informed by the Councilor of the ongoing affairs of the State Medical Association.

Respectfully submitted,
G. Robert Bartron, M.D.
Councilor, Second District

The Reference Committee reviewed the report of the Councilor from the Second District Medical Society and recommended the report be accepted as submitted.

REPORT OF THE THIRD DISTRICT COUNCILOR

As Councilor of the Third District Medical Society, I am re-

porting on the following meetings held in our district during the past year:

- July 14, 1980: There was a scientific program by Dr. Rod Parry from the Department of Pulmonary Medicine, University of South Dakota Medical School. Dr. Curtis Wait, Brookings, volunteered to accept a position on the First Planning District, Sub-area Council of Health.
- August 21, 1980: Dr. J. A. Muggly, Madison, was presented a plaque in appreciation for his services rendered in establishing PSRO in the state of South Dakota be Paul Jensen of the South Dakota PSRO. A scientific presentation on intraepithelial neoplasia of the cervix was made by Dr. Samir Abu-Ghazaleh, Yankton, South Dakota.
- October 17, 1980: Dr. Winston Odland, Aberdeen, made the annual presidential visit to the district. He discussed numerous topics including the delivery of health care, maintenance of ethical standards under increasing pressure from bureaucratic federal legislation, and National Health Insurance. Dr. Saul Friefeld, Brookings, is to be presented by the district for the Distinguished Service Award and Dr. J. A. Muggly to be presented for the Community Service Award.
- December 11, 1980: Annual Meeting: Officers for the new year are Dr. Wait, Brookings, President; Dr. Appelwick, Madison, Vice President; Dr. Bandiera, Brookings, Secretary-Treasurer. Scientific program on oncology was presented by Dr. Steven Johnson and Dr. Loren Tschetter of the Central Plains Clinic, Sioux Falls.
- February 19, 1981: Scientific program was presented by Dr. Raszowski, Department of Gastroenterology, University of South Dakota Medical School. Arthur Lampert, M.D. was renominated as Councilor; Dr. Curtis Wait, Alternate Councilor; Delegates to the annual meeting, Dr. Richard Wake and Dr. Curtis Wait.

Respectfully submitted,
Arthur A. Lampert, Jr., M.D.
Councilor, Third District

The Reference Committee reviewed the report of the Councilor from the Third District Medical Society and recommended it be accepted as submitted.

REPORT OF THE FOURTH DISTRICT COUNCILOR

The annual meeting of the Fourth District Medical Society was held on January 12, 1981. At this time, election of officers was as follows: President, Lester Krawitt, M.D.; Vice President, Phillip Hoffsten, M.D.; and Secretary-Treasurer, M. R. Cosand, M.D. Dr. John Davis was elected as Delegate and Dr. Lester Krawitt as Alternate Delegate.

At this meeting, SDSMA President, Dr. Winston Odland, and Executive Secretary, Robert Johnson, made their annual visitation. The program consisted of a discussion of the issues before the upcoming legislative session.

As in previous years, the entire district participated in Athletic Physical Clinics, one in May and the other in September, for all Pierre and Ft. Pierre athletes.

The following CME programs were sponsored in cooperation with the In-Service Department at St. Mary's Hospital:

- January 10, 1980 "Basic CPR Certification"
- February 7, 1980 "Seizures in Children"
Allen Kelts, M.D.
- March 18, 1980 "Psychotropic Medications"
Charles Lord, M.D.

- April 22, 23, 1980 "Neonatology"
Larry Fenton, M.D.
- May 27, 1980 "Cardiac Rehabilitation"
M. Gilliland, R.N., J. Langdon, M.D.
- June 12, 1980 "Medical Evaluation of the Pediatric Patient with Handicaps"
Allen Kelts, M.D.
- July 15, 1980 "Fluids and Electrolytes—Acid Base Balance"
Phillip Hoffsten, M.D.
- September 16, 1980 "Emergency Medicine"
Howard Burns, M.D., Charles Hollerman, M.D.
- October 23, 24, 1980 "Lake Sharpe Cardiology Seminar"
Doctors Bessinger, Kiser, Rogers, VanTassel and VanSon
- November 18, 1980 "Infectious Diseases"
Charles Kallick, M.D.

Respectfully submitted,
R. C. Jahraus, M.D.
Councilor, Fourth District

The Reference Committee reviewed the report of the Councilor from the Fourth District Medical Society and recommended it be accepted as submitted.

REPORT OF THE FIFTH DISTRICT COUNCILOR

The Huron District 1980 year began with the March 27, 1980 meeting at the Legion Club with a steak dinner planned by our able Secretary, Emil Hofer, M.D. It was a stormy night, and so our planned program was cancelled. Reports of officers and the councilor filled the void.

On May 22, we held our pre-convention meeting to instruct the delegates on their duties and voting for the Aberdeen meeting. The councilor and delegates attended the State Meeting in June of 1980 in full force.

The councilor attended the September 12 meeting of the Council in Sioux Falls, South Dakota.

Dr. Robert Schroeder of Miller, South Dakota was our newest member inducted in 1980 with the opening of our September 25 meeting date at the Legion Club once again. The State President, Winston Odland, M.D., of Aberdeen brought the group up to date on meetings with the Governor and the South Dakota Department of Health in Pierre, South Dakota.

The district councilor and Dr. Saylor attended the Council meeting in Sioux Falls on November 22.

On December 2, 1980, we had a program consisting of a talk on coronary artery surgery by Dr. Robert Willix of Sioux Falls. New officers were elected: President, Robert Hohm, M.D.; Vice President, George Nicholas, M.D.; Secretary, Emil Hofer, M.D.; and Board of Censors, William Hanson, M.D.

The March 26, 1981 meeting featured the Medical School, and again was held at the Legion Club with a steak dinner.

Respectfully submitted,
David Buchanan, M.D.
Councilor, Fifth District

The Reference Committee reviewed the report of the Councilor from the Fifth District Medical Society and recommended acceptance of this report.

REPORT OF THE SIXTH DISTRICT COUNCILOR

The Sixth District has been growing at a rapid rate with the addition of many physicians in the specialties. We have had only two meetings this year; those being the visit of our President, Dr. Winston B. Odland, and the election of officers for the coming year. The officers for the coming year are: President, Dr. Charles Monson, Parkston; Secretary, Dr. Richard Hockett, Mitchell; Delegates, Dr. Walter P. Baas and Dr. Michael Haley, Mitchell; Alternate Delegates, Dr. Charles Monson and Dr. John McCann, Parkston; Councilor, Dr. Richard Gere, Mitchell.

It is hoped that with the increase in the number of physicians in the District, and the fact that many of them are young, we

can become very active.

Respectfully submitted,
Richard G. Gere, M.D.
Councilor, Sixth District

The Reference Committee reviewed the report from the Councilor for the Sixth District Medical Society and recommended acceptance of his report.

REPORT OF THE SEVENTH DISTRICT COUNCILOR

The Seventh District Medical Society met regularly on the first Tuesday of each month during the past year with the exception of June, July and August. The business conducted during the early part of the year involved the city of Sioux Falls Urban Health Grant and its possible relationship with the Family Practice Residency. Attempts were made by a committee from the Seventh District to coordinate the two programs in such a manner to benefit each other; however, this did not succeed and has not developed to this point.

Mr. Ed Zulk presented a program to the Seventh District explaining the concept and function of his grant and how it would be used for health care in the city of Sioux Falls.

During the year, the Seventh District established various positions to include: 1) Opposition to a full time city physician; 2) Not sanctioning the Nutri/System Diet Program; 3) Support Family Practice Residency Program in staffing of the City Health Department.

The Seventh District continues to grow with the addition of new physician members throughout the year.

In September, Dr. Winston Odland, President of the South Dakota State Medical Association, presented a program and report of the House of Delegates meeting of the American Medical Association and discussed the ongoing problems of the Medical Association.

Dr. Charles Hollerman was a guest at our meeting in October and discussed the present status of the Medical School.

Dr. Lawrence Finney was elected President of the Society and assumed his duties in January of 1981.

Respectfully submitted,
Larry L. Sittner, M.D.
Councilor, Seventh District

The Reference Committee reviewed the Councilors' report from the Seventh District Medical Society and recommended acceptance of this report.

REPORT OF THE EIGHTH DISTRICT COUNCILOR

The Eighth District Medical Society met on four occasions during the past year.

The first meeting was April 16, 1980. At this meeting, business discussion was centered primarily around topics coming before the annual meeting of the South Dakota State Medical Association.

The second meeting was held July 29, 1980. Dr. Samir Z. Abu-Ghazaleh was accepted into the District Society. \$100 was appropriated to the educational fund of SoDaPAC. The scientific meeting was then conducted by Dr. Gordon Held on the epidemiology of lung cancer.

The third meeting was held in Vermillion on October 14, 1980. Since there was inadequate numbers of persons present for a quorum, the business meeting was deferred. A scientific meeting was presented by the University of South Dakota Psychology Department dealing with family and marital counseling.

The fourth meeting was held January 27, 1981 at the Sheraton Motor Inn in Yankton. Due to a recent illness of Dr. Hal Fletcher, Dr. Phil Blum has taken over as acting President. At this meeting, Dr. Charles Hollerman gave a report on the status of the Medical School. Applications were received for membership into the District Society from Dr. Beth Johnson, Dr. John Sternquist and Dr. John Jacobsen. Dr. Richard Brinkman was nominated and elected as the new acting Vice President. A finance committee was appointed to review the funds in the Eighth District treasury and make recommendations as to their disposal. Delegates for the state convention were selected and include Dr. Jay Hubner, Dr. Thomas Olson and Dr. Richard Porter. Alternates were Dr. Richard Thornton, Dr. Tom Johnson and Dr. Malcolm Jameson.

Dr. Winston Odland, President of the State Medical Association, was present and spoke to the Eighth District Medical Society on national and state medical business.

Respectfully submitted,
Frank D. Messner, M.D.
Councilor, Eighth District

The Reference Committee reviewed the report of the Councilors' for the Eighth District Medical Society and recommended the acceptance of this report.

REPORT OF THE NINTH DISTRICT COUNCILOR

The Black Hills District Medical Society held six business meetings during the year at which routine business was conducted.

At the February 17 meeting, we increased our district dues from \$20 to \$40 per year. Winston Odland, M.D., President, made his presidential visitation at our November 13 meeting. He was accompanied by Robert Johnson, Executive Secretary. Charles Hollerman, M.D., Dean of the USD School of Medicine, was present at our April 14 meeting and discussed recent developments at the Medical School.

Our new officers for the year 1981 are:

N. R. Whitney, M.D., President

R. D. Bloemendaal, M.D., Vice President

A. J. Barrett, M.D., Secretary-Treasurer

Roger Millea, M.D. was voted to retain his chair as Councilor for a three year term.

Ed James, M.D. was named to serve as Alternate Councilor.

Our other Councilors, A. J. Barrett, M.D. and Robert Ferrell, M.D. continue to serve as well as Alternate Councilor, N. R. Whitney, M.D.

Respectfully submitted,
A. J. Barrett, M.D.
Councilor, Ninth District

The Reference Committee reviewed the Councilors' report from the Ninth District Medical Society and recommended acceptance of this report.

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REPORT OF THE ELEVENTH DISTRICT COUNCILOR

The Northwest District Medical Society held four regular business meetings during the course of the year excluding the summer months. These meetings were held in October, November, January and February, and all meetings featured a guest speaker for a scientific program. Topics covered an antibiotic review sponsored by Merck, Sharp & Dohme, recent advances in otology and neck mass biopsy, a discussion about coronary artery spasm and cardiovascular surgery in coronary artery bypass grafts. The January 1981 meeting was in conjunction with the visitation by the President of the South Dakota State Medical Association including Executive Secretary, Bob Johnson, and Kevin Loge.

The Eleventh District lost one physician during the past year when Dr. James Ryan moved to Minot, North Dakota to assume a position with the University of North Dakota School of Medicine.

Officers elected for 1981 are: James Collins, M.D., President; Morris Benson, M.D., Vice President; Leonard Linde, M.D., Secretary; and James Collins, M.D., Delegate.

Respectfully submitted,
James F. Wunder, M.D.
Councilor, Eleventh District

The Reference Committee reviewed the report of the Councilor from the Eleventh District Medical Society and recommended the acceptance of this report.

REPORT OF THE TWELFTH DISTRICT COUNCILOR

The Whetstone Valley District Medical Society met on June 18, 1980 at the home of Dr. Joseph Kass, Rosholt. This was the annual visit by President, Winston B. Odland, M.D., and Executive Secretary, Robert Johnson. Dr. Ed Johnson was designated as the district representative for the centennial activities. Dr. Joseph Kass was elected President and Dr. David Oey Secretary.

The Whetstone Valley District Medical Society met on November 12, 1980 in Webster. The speaker was Dr. Lloyd E. Solberg. His topic was "Cardiac M-mode Echocardiography."

Respectfully submitted,
Eldon E. Bell, M.D.
Councilor, Twelfth District

The Reference Committee reviewed the report from the Councilor from the Twelfth District Medical Society and recommended the acceptance of this report.

REPORT OF THE COMMISSION ON LEGISLATION & GOVERNMENTAL RELATIONS

The Commission met twice during this past year; the first meeting being held on Thursday, September 11, 1980.

The Commission reviewed three state legislative bills which addressed minimum standards and disclosure requirements for health insurance policies sold. Although accepted for information only, the Commission agreed with the concept of minimum standards.

Physical exams for junior/senior high school student athletes was discussed, and an oral report was presented by the subcommittee chairman. A final report by the subcommittee at the next meeting was requested by the Commission members.

The Commission received a resolution from the South Dakota Pharmaceutical Association to support a bill ending free sampling of legend drugs in South Dakota by manufacturers; however, the Commission members felt that the elderly and indigent would suffer, that the distribution of state medicaid dollars would shift, and that samples used on a trial basis curtail prescription cost to the consumer. The Commission recommended that the State Medical Association support limited sampling of prescription drugs at physician's request.

Information on the meeting between Governor Janklow, Dr. Winston Odland and Mr. Robert Johnson was presented to the Commission. In addition, Mr. Johnson informed the Commission that the South Dakota State Board of Medical and Osteopathic Examiners has directed the State Medical Association staff to revise the physician assistant statutes. This was accepted for information only.

The 1981 legislative program was discussed by the Commission and would be finalized at the next meeting.

It was brought to the attention of the Commission that prior to entering college at SDSU and USD, nineteen and twenty year old females must receive a rubella vaccine. The Commission referred this matter to the Commission on Scientific Medicine.

The second meeting of the Commission was held on Wednesday, January 7, 1981.

The Commission again discussed policy for student athletic physical exams and reviewed the initial and interim physical health forms which were presented by the subcommittee chairman. The Commission recommended to the Council the acceptance of both questionnaires, and that if passed by the House of Delegates, the questionnaires be personally delivered to the South Dakota High School Athletic Commission for their review and consideration.

The Executive Secretary relayed to the Commission members that provisions in the proposed Certificate of Need legislation to be introduced at the 1981 legislature by the State Department of Health concerning physicians' offices and purchases of major medical equipment have been deleted from the legislation. The Commission recommended that the State Medical Association retain its position on Certificate of Need and oppose any extension of any Certificate of Need to physicians' offices.

A proposal for the 1981 legislature "Make South Dakota Safe!" was submitted by the pediatricians in South Dakota to the Commission requesting that a resolution be passed supporting actions which would reduce unnecessary deaths on South Dakota highways. The Commission tabled the request; however, the Commission agreed with the principles and concepts but had reservations on mandating the request into law.

The Commission received a letter requesting the State Medical Association's support for a bill requesting funds to remodel a major building structure at the Human Services Center in Yankton. The Commission recommended to the Executive Commission that the State Medical Association support the request.

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The Medical and Hospital Associations' Commission on Legislation then jointly met and discussed common 1981 legislative concerns. Both Commissions expressed concern on the professional qualifications of registered nurse members on the South Dakota Board of Nursing. The Commission recommended that the State Medical Association jointly introduce legislation with the South Dakota Hospital Association to remove from SDCL 36-9-7 the professional qualifications of registered nurse members for the South Dakota Board of Nursing, and further remove restrictions on the nominating process for nurse members. The Commission also recommended that the State Medical Association endorse the Hospital Association's action to repeal SDCL 34-8-8.

The following is the finalized 1981 legislative program adopted by the Commission:

Sponsored Bills

1. Repeal statute that prohibits the Board of Trustees of a county hospital from discriminating between practitioners of the various healing arts.
2. Repeal statute that requires physicians to record their license in the county where they reside and are engaged in practice.
3. Delete present qualifications of registered nurses and give the Governor more latitude in appointing to the Board of Nursing.

Endorsed Bills

1. Renovate areas of the medical institute building at the South Dakota Human Services Center.

Opposed Bills

1. Require disclosure of test questions, the subjects answer sheet and copy of correct answer sheet to the Medical College Admission Test.
2. Prohibits electro-shock treatments on patients who do not grant their voluntary consent.

Respectfully submitted,
Stephen N. Haas, M.D.

Chairman
Commission on Legislation and
Governmental Relations

The Reference Committee carefully reviewed the report of the Commission on Legislation and Governmental Relations. The Reference Committee recommends the acceptance of the report of the Commission on Legislation and Governmental Relations and would like to commend the Commission for their work during the year.

REPORT OF THE COMMISSION ON MEDICAL SERVICE

The Commission on Medical Service met twice during the past year; on September 11, 1980 and on March 28, 1981 in Sioux Falls.

The Commission received a report from Dr. C. D. Monson on the AMA Rural Health Conference which he attended in Boston, Massachusetts on April 17, 18, 1980. The AMA cancelled the 1981 conference because of poor attendance in 1980.

The Commission reviewed information on the American Society of Anesthesiologists' Relative Value guide and referred this item to the Long Range Planning Committee for their review and consideration.

Statistical information on the numbers of physicians, physician assistants, nurse practitioners and nurse midwives was provided to the Commission. This data covered the last six years and indicated a considerable increase in health providers in South Dakota.

The Commission reviewed the AMA guidelines for the categorization of hospital emergency capabilities and recommended that the Council not accept the AMA draft because it is not applicable to South Dakota; however, if in the future a program is drafted that better suits South Dakota, the SDSMA would be interested in reviewing such a proposal.

The Commission reviewed information on the National Health Service Corp program in South Dakota, including the numbers of personnel in South Dakota, their placement and the monies expended for this program. This Commission shall continue to monitor this program in South Dakota.

A request for endorsement was received from Medical Data

Banc which provides a health information card and central information services for those subscribing to their service. The Commission recommended to the Council that the SDSMA not endorse this program.

The Commission reviewed forms proposed for use by the State Health Department's Family Planning Program and expressed their concerns and feelings that the form as proposed should not be utilized. The Health Department concurred with the Commission and indicated the form would not be utilized and that any future proposals would be submitted to the SDSMA for review and approval prior to use.

Dr. Saylor reported that he met with the Watertown District Medical Society to discuss the Emergency Medical Service Northeast Regional Proposal; however, the district took no action regarding implementation of the proposal.

Pioneer Hi-Bred International corresponded with a Commission member, Dr. Saylor, concerning their health screening program for South Dakota. The Commission reviewed Dr. Saylor's response outlining his concerns for such a program, concurred with that response and recommended that if Pioneer Hi-Bred proceeds with this screening program, perhaps they should modify it to stress basic health concepts such as weight reduction, smoking or hypertension.

The Commission reviewed a request from the Southeastern Mental Health Center in Sioux Falls for a letter of support to accompany their grant request. The Commission recommended to the Council that the SDSMA support the grant request for the Southeastern Mental Health Center.

Respectfully submitted,

C. D. Monson, M.D.

Chairman

Commission on Medical Service

The Reference Committee reviewed the report of the Commission on Medical Service. The Reference Committee recommends the acceptance of the report of the Commission on Medical Service and would like to commend the Commission for their work during the year.

REPORT OF THE COMMISSION ON SCIENTIFIC MEDICINE

The Commission on Scientific Medicine met in August 1980 to deal with problems that were forwarded from the Council for their evaluation and to plan the 1981 centennial meeting of the South Dakota State Medical Association. The first of these meetings occurred on August 9, 1980.

The Premarital Serology Testing, which was forwarded from the Council to the Commission on Scientific Medicine, was dealt with by recommending to the Commission on Legislation and Governmental Affairs that the Premarital Serology Testing law be rescinded because this test is not cost effective, the incidence of new positive cases is extremely low, and there are other more effective avenues of education and screening.

A problem which had been referred from the Council with regard to the recommendations of the State Health Department with regard to Ophthalmia Neonatorum Prophylaxis was discussed. The Commission reaffirmed the recommendation of the Center for Disease Control that any of the three treatment modes, Ag NO3, Tetracycline and Erythromycin, are appropriate for the prophylaxis of ophthalmia neonatorum, and that one should not be determined preferable over the other two.

The remainder of the meeting was dedicated to the planning of the 1981 Centennial Meeting of the South Dakota State Medical Association. It is the hope of the Commission that the program meets with the approval of the membership on the occasion of our 100th meeting.

Respectfully submitted,

James C. Larson, M.D.

Chairman

Commission on Scientific Medicine

The Reference Committee reviewed the report of the Commission on Scientific Medicine. The Reference Committee recommends acceptance of this report. The Reference Committee wishes to commend the members of the Commission for their diligent efforts throughout the past year on behalf of the members of the State Association.

REPORT OF THE COMMISSION ON INTERNAL AFFAIRS, COMMUNICATIONS AND LIAISON

The Commission met twice during the year. These meetings were held in Sioux Falls.

The Commission discussed the grant scholarships to be provided by the former Health Career Loan Fund; Dr. William Quick volunteered to be the representative from the Commission to work with the Auxiliary members on the program; this work was carried out with great expediency. The grant program is functional and ready for applications at this point.

Dr. Allan Hartzell's recommendations for an organization and recruitment program for membership in the State Association was reviewed, and it was moved by the Commission members that the report be sent to the Council for its review.

It was recommended to the Council that they review State Medical Association dues for the various physicians' groups within the state in an attempt to gain more physician members in the State Association.

It was recommended also that the Prseident of each District Medical Society be contacted regarding appointing a physician responder who would develop good rapport with the public media regarding news stories, radio and TV coverage. It was also recommended that this Commission develop a code of cooperation setting forth mutual obligations and responsibilities of the media and the health providers.

A discount proposal for car rental by the Hertz Corporation was reviewed by the Commission, and it was recommended to the Council that this program be accepted.

Copies of codes of cooperation from various states were reviewed by the Commission, and it was recommended to the Council that the South Dakota State Medical Association revise its present code of cooperation and that a committee be formed from members of the Commission to study the information obtained and bring it back to the Commission that the same committee be authorized to develop guidelines and set up recommendations regarding telephone calls and requests for release of information from physicians' offices by members of the Bar Association.

The members of the Commission met with two representatives of the South Dakota State Bar Association regarding a possible seminar to be held jointly in the spring of 1982 and recommended that the proposal be referred to the Commission on Scientific Medicine for further consideration.

Patient information brochures regarding health maintenance organizations were reviewed, and it was moved that further discussion of this issue be deferred until it is determined what the government's guidelines might be regarding HMO's in the future.

A proposal by Prairie States Life Insurance Company regarding a proposed disability income policy was reviewed, and it was decided to make no recommendations on this proposal to the Council.

Reports were submitted to the Council during the spring meeting regarding the physician recruitment program and the public information program. Both of these were received for information only.

During the past year, the following South Dakota physicians have died:

William Sercl, M.D.	Sioux Falls
W. L. Spain, M.D.	Rapid City
Claude Delaney, M.D.	Sturgis
R. V. Avotins, M.D.	Faulkton
Fred Leigh, M.D.	Huron

The Health Career Loan Fund reported the following activity during the past twelve months:

Balance in Savings Account	
March 1, 1980	\$ 6,242.62
Income	
Interest	\$ 893.98
Principal	2,294.67
Total Income	\$3,188.65
	<u>3,188.65</u>
	\$ 9,431.27
Expenses	
Loans (7)	3,500.00
	<u>3,500.00</u>
	\$ 5,931.27

Balance in Savings Account	
March 1, 1981	\$ 5,931.27
Assets	
Money Market Certificates	\$11,046.20
Savings Account	5,931.27
Outstanding Loans	15,009.55
Total Assets	<u>\$31,987.02</u>

During the year, the Commission has reviewed each financial report of the State Medical Association, the general account and the building fund. The Budget and Audit Committee, consisting of the Executive Committee and Chairman of this Commission, considered and reviewed a budget for the fiscal year 1981-82, and it was submitted to the Council for its consideration and transmittal to the House of Delegates. The proposed budget is attached as part of this report.

Respectfully submitted,
Lawrence W. Finney, M.D.
Chairman
Committee on Communications,
Internal Affairs and Liaison

The Reference Committee reviewed the report of the Commission on Internal Affairs, Communications and Liaison. The Reference Committee recommends the acceptance of this report. The Reference Committee wishes to commend the members of the Commission for their diligent efforts throughout the past year on behalf of the members of the State Association.

**PROPOSED BUDGET 1981-1982
SOUTH DAKOTA STATE MEDICAL ASSOCIATION**

**GENERAL FUND
INCOME**

ITEM	BUDGETED 80-81	PROPOSED 81-82
State Dues	\$150,000.00	\$165,000.00
Annual Meeting	15,000.00	16,000.00
Refunds	4,000.00	4,000.00
Car Reimbursement	252.00	252.00
Continuing Med. Ed.	1,000.00	750.00
Salary Rcimbursement	9,300.00	11,700.00
Other Programs		
7th Dist. Salary Reim.	1,350.00	1,350.00
Equip. Repl. Fund	2,400.00	2,400.00
AAFP Salary Reimb.	1,500.00	1,500.00
Medical Student Dues	---	1,000.00
Collection Service	2,000.00	2,825.00
Interest	6,500.00	10,000.00
	<u>\$193,302.00</u>	<u>\$216,777.00</u>

EXPENSES

ITEM	BUDGETED 80-81	PROPOSED 81-82
Salaries	\$90,000.00	\$101,700.00
Social Security	6,000.00	6,800.00
Legal & Audit	6,000.00	6,500.00
Telephone	3,500.00	3,700.00
Office Supplies	6,500.00	7,300.00
Dues & Subscriptions	900.00	900.00
Physicians' Travel	9,000.00	11,000.00
Annual Meeting	15,000.00	16,000.00
Public Relations	4,000.00	4,000.00
Journal Subsidy	5,000.00	6,000.00
Postage	5,000.00	5,500.00
Miscellaneous	100.00	100.00
Legislation	5,500.00	5,500.00
Car Expenses	1,200.00	1,500.00
Staff Travel	10,000.00	11,000.00
Insurance	1,200.00	1,500.00
Retire/Fringe Benefits	16,000.00	18,500.00
Taxes	200.00	---
Auxiliary Newsletter	800.00	1,000.00
Employment Tax	400.00	500.00
Continuing Med. Ed.	800.00	750.00

Income Tax	1,000.00	500.00
Repl. of Equipment	2,000.00	1,000.00
Med. Student Fund	---	1,000.00
	<u>\$190,100.00</u>	<u>\$212,250.00</u>
Reserve	3,202.00	4,527.00
	<u>\$193,302.00</u>	<u>\$216,777.00</u>

BUILDING FUND INCOME

ITEM	BUDGETED 80-81	PROPOSED 81-82
Foundation Rent	\$11,880.00	\$12,780.00
Bd. of Exam. Rent	2,400.00	3,600.00
Interest Income	<u>7,600.00</u>	<u>10,000.00</u>
	<u>\$21,880.00</u>	<u>\$26,380.00</u>

EXPENSES

Salaries, Staff	\$10,580.00	\$14,425.00
Utilities	2,500.00	2,825.00
Taxes & Insurance	4,500.00	5,000.00
Maintenance & Supplies	3,200.00	2,780.00
Legal & Audit	<u>1,100.00</u>	<u>1,350.00</u>
	<u>\$21,880.00</u>	<u>\$26,380.00</u>

JOURNAL INCOME

ITEM	BUDGETED 80-81	PROPOSED 81-82
Advertising	\$18,000.00	\$18,500.00
Subscriptions	800.00	1,000.00
Refunds	720.00	720.00
Journal Subsidy	5,000.00	6,000.00
Miscellaneous	<u>600.00</u>	<u>600.00</u>
	<u>\$25,120.00</u>	<u>\$26,820.00</u>

EXPENSES

ITEM	BUDGETED 80-81	PROPOSED 81-82
Salaries	\$ 2,220.00	\$ 2,220.00
Legal & Audit	100.00	100.00
Social Security	100.00	100.00
Telephone	100.00	115.00
Postage	1,000.00	1,500.00
Office Supp. & Print.	<u>21,600.00</u>	<u>22,785.00</u>
	<u>\$25,120.00</u>	<u>\$26,820.00</u>

REPORT OF THE COMMISSION ON PROFESSIONAL LIABILITY

The Commission met once during the past year in Sioux Falls, South Dakota. At that meeting, representatives of American Health Systems, San Francisco, provided information on the Minnesota Medical Insurance Exchange and proposed an actuarial study to determine if it would be advantageous to have MMIE market professional liability insurance in South Dakota.

Representatives of the St. Paul Companies also met with the Commission and discussed South Dakota premium rates and loss ratios as well as loss prevention programs.

A discussion was held concerning the duties and parameters of this Commission. This subject will be explored further during the coming year. The Commission will also present a recommendation concerning the proposal from American Health Systems after further study has been completed.

Respectfully submitted,
Michael Rost, M.D.
Chairman

Commission on Professional Liability

The Reference Committee reviewed the report of the Commission on Professional Liability. The Reference Committee recommends acceptance of this report. The Reference Committee wishes to commend the members of the Commission for their diligent efforts throughout the year on behalf of the members of the State Association.

REPORT OF THE GRIEVANCE COMMISSION

The Grievance Commission has met on two occasions during the past year and will meet again at the time of the Annual Meeting. There have been an abundant number of rather complicated cases presented to the Grievance Commission. It is felt by the Chairman of the Grievance Commission that a careful delineation of the functions and duties of the Grievance Commission needs to be determined and evaluated so that their complete function will be more fully understood by the present bylaws which state only briefly what the functions of this commission should be.

It is hoped that in the ensuing year the Grievance Commission and/or some committee from the State Medical Association can help to devise such a determination of function of this very important public relations committee.

I would most particularly like to thank the staff of the State Medical Association for all the tremendous help which has been given to the Grievance Commission during the past year as well as to thank all members of the Grievance Commission for their support.

Respectfully submitted,
Gerald E. Tracy, M.D.
Chairman

Grievance Commission

The Reference Committee reviewed the report of the Grievance Commission and recommends acceptance of this report.

REPORT OF THE LONG RANGE PLANNING COMMITTEE

Members of the committee are: T. H. Sattler, M.D., Chairman; Karl Wegner, M.D.; Dennis Johnson, M.D.; Michael Pekas, M.D.; Charles Tesar, M.D.; H. J. Stensrud, M.D.; W. Nicol Guddal, M.D.; and Stanley Altman, M.D.

The committee met on August 19, 1980 and December 9, 1980.

Prime area of study and monitoring continues to be the South Dakota health planning programs. Physician members of the South Dakota State Medical Association have contributed a vital and critical input into monitoring as well as developing effective elements of the State Health Plan. The newer area of "appropriateness of review" has been of particular interest and will require continuing dedicated monitoring commitment by our members.

Major changes in health planning will certainly evolve due to funding program changes by the new administration in Washington.

What type, if any health planning, will survive or replace present programs is uncertain at this time. The "pro-competition" proposals warrant very serious short and long term evaluation. The effects on medical care service could be profound and warrants in-depth thoughtful assessment.

Complete reports are on file with the Council and the South Dakota State Medical Association office.

Respectfully submitted,
T. H. Sattler, M.D.
Chairman

Long Range Planning Committee

The Reference Committee reviewed the report of the Long Range Planning Committee and recommends acceptance of the report.

REPORT OF THE SOUTH DAKOTA POLITICAL ACTION COMMITTEE

1980 produced some pleasing and some not so pleasing results for SoDaPAC.

Since this was an election year, we expended our accumulated monies in supporting 61 legislative candidates. 46 of these won—76%. I know that this made the job of our representatives in Pierre less arduous.

We were pleased to receive a total of \$21,000 from AMPAC in support of several candidates for U.S. Congress.

Despite these positive elements, it is sad to say that less than 25% of South Dakota physicians supported SoDaPAC in its efforts. This is especially difficult to understand when we consider the regular dues for SoDaPAC-AMPAC were only \$30 and sus-

taining only \$100 from one of the highest income groups in society!—one which is threatened with the loss of so much through political action.

I can only hope that we can get more participation in our continued efforts in the behalf of private medicine.

All of you can help. Join us.

Respectfully submitted,
T. J. Wrage, Jr., M.D.
Chairman
SoDaPAC

The Reference Committee reviewed the report of the South Dakota Political Action Committee and recommends acceptance of the report.

REPORT OF THE ENDOWMENT ASSOCIATION

The South Dakota Medical School Endowment Association Committee met at the time of the annual meeting and has not met since that time. However, there have been telephone conversations and the affairs of the Endowment Association have been conducted in this manner. The Endowment Association continues to have as its primary function the loaning of funds to medical students through either matching programs or directly. These are monitored at the University and monitored also by members of the Endowment Association Committee.

There also has been monies expended in support of the development of the South Dakota Medical School Alumni Association, and these were committed for a three year period of time. Following this year, one more year of support will be given in that direction. Following this, it is anticipated that the Alumni Association can stand on its own, but certainly the Endowment Association will cease with its support at that time. The committee, in general, wishes to thank all of the physicians of the State of South Dakota as well as other generous contributors for their contributions to a most worthwhile and important project. The Endowment Association uses most of the contributions providing loans for students who otherwise cannot receive funds for their medical education.

Respectfully submitted,
Gerald E. Tracy, M.D.
Chairman
Endowment Association

The Reference Committee reviewed the report of the Endowment Association and recommends acceptance of the report.

REPORT OF THE COMMITTEE FOR CONTINUING MEDICAL EDUCATION

No specific activity of the committee was performed this year. The organization for continuing medical education now basically has been taken over by the American Medical Association to the exclusion of the LCCME.

It is anticipated that a spring or summer meeting will be held by our committee to discuss this with a report hopefully from Dr. Quinn, who has been active on a national level in this problem.

Respectfully submitted,
K. G. Koob, M.D.
Chairman

Committee for Continuing Medical Education

The Reference Committee reviewed the report of the Committee for Continuing Medical Education and recommends acceptance of the report.

ANNUAL MEETING MINUTES SOUTH DAKOTA MEDICAL SERVICE, INC. CORPORATE BODY MEETING

May 29, 1981, 9:30 a.m.
Ramada Inn, Sioux Falls, South Dakota

Chairman Howe called the meeting of the Corporate Body of South Dakota Medical Service, Inc., to order at 9:30 a.m. on May 29, 1981, at the Ramada Inn, in Sioux Falls, South Dakota.

Upon roll call, the following members of the Corporate Body of the South Dakota Medical Service, Inc., were present:

Doctors Winston Odland, Bruce Lushbough, Durward Lang, Joseph Hamm, Duane Reaney, Howard Saylor, Gerald Tracy, Russell Harris, G. Robert Bartron, A. A. Lampert, Jr., R. C. Jahraus, David Buchanan, R. G. Gere, William O. Rossing, Richard Gunnarson, Guy Tam, Larry Sittner, Gordon Held, Frank Messner, N. R. Whitney, Robert L. Ferrell, A. J. Barrett, M. George Thompson, James Wunder, Eldon Bell, David Seaman, John Christopher, Karlis Zvejnieks, James Rud, James Larson, Richard Wake, Curtis Wait, John David, Robert Hohm, Louis Karlén, Chris Moller, Maynard Porter, John Ochsner, Gail Benson, Robert Talley, Neil Elkjer, Daniel Kennelly, Gene Koob, Lynn Henrickson, Dennis Johnson, R. I. Porter, Jay Hubner, Tom Olson, O. Myron Jerde, Thomas Kraska, Charles Tesar, Robert Westaby, Charles Loos, Louis Hogrefe, James Collins, E. A. Johnson, and students Daryls Hofer and Dave Faragher.

A quorum being present, the Chairman declared the Annual Meeting of the Membership of the Corporate Body of South Dakota Medical Service, Inc., to be duly in session for the transaction of business.

Dr. James Larson moved that reading of the minutes of the last meeting of the Corporate Body, being the 1980 Annual Meeting, be waived, the same having been published in the Corporate Handbook and previously mailed to each member. Such motion was seconded by Dr. Tracy. Upon voice vote, the same was approved unanimously.

The Chairman of the Board, Don Howe, presented his message to the Corporate Body as contained in writing in the Delegate Handbook. He reviewed the decision of Blue Shield to not continue as Medicare Part B Carrier for the State of South Dakota. The primary reason for such an election was the insistence of the federal government that Blue Shield use corporate funds to subsidize the program. He reviewed a number of problems facing medical service plans including mandated benefits by the State Legislature.

He thanked the physicians who support South Dakota Blue Shield and recognized past and present members of the Board of Directors.

Mr. Erickson presented the Financial Report for the year 1980. Blue Shield paid out 90.6% of all premium dollars on claims during 1980. As a result, it had a substantial underwriting loss, which, after deducting investment income, resulted in an actual 1980 loss of \$126,000.

Dr. Harris moved approval of the Financial Report. Such motion was seconded by Dr. Odland. Upon voice vote, the same was approved unanimously.

The Chairman called for the report of the Nominating Committee. The Chairman of the Nominating Committee, Dr. James Collins, submitted the following persons' names for nomination for re-election to the Board:

Robert Bloemendaal of Rapid City,
Roscoe Dean of Wessington Springs,
James Jelbert of Spearfish.

He reported that all three of the persons nominated are eligible for a three-year term.

The Chairman called for nomination by members of the Corporate Body from the floor for offices of Blue Shield directors. No nominations were made from the Body.

Dr. James Larson moved that the nominations cease and that the Secretary be instructed to cast a unanimous ballot for the nominees, namely, Robert Bloemendaal, M.D., Roscoe Dean, M.D., and James Jelbert. Such motion was seconded by Dr. Bartron. Upon voice vote, such motion was approved unanimously.

The Chairman called for consideration of other Old Business.

Dr. Hamm asked if Blue Shield lost money in 1979. Mr. Erickson stated that in 1979 Blue Shield had a \$487,000 gain. He stated that so far in 1981, Blue Shield has sustained substantial losses but hopes to offset these with premium increases.

Dr. Bartron asked Mr. Erickson if Blue Shield lost any business by groups changing to self insurance. Mr. Erickson stated Blue Shield had lost one or two groups and he anticipated losing additional groups. He stated there are predictions that as many as 40% of businesses carrying health insurance plans will change to self insurance. Blue Shield is offering to provide administrative

services for such self insuring businesses.

Chairman Howe thanked the Corporate Body for their co-operation on Blue Shield programs throughout the past year and the assistance that they have always given South Dakota Blue Shield.

The Chairman asked if there was any further business anyone cared to present. There being no further business, Dr. G. Robert Bartron moved that the meeting be adjourned. Such motion was seconded by Dr. Duane Reaney. Upon voice vote, the same was approved unanimously.

The meeting was duly adjourned at 10:00 a.m.

John H. Zimmer
Secretary

PRESIDENTIAL OATH OF OFFICE

I SOLEMNLY SWEAR THAT I shall carry out the duties of the President of the South Dakota State Medical Association to the best of my ability. I shall strive constantly to maintain the ethics of the medical profession and to promote the public health and welfare. I shall dedicate myself and my office to improving health standards and to the task of bringing increasingly improved medical care to the people of South Dakota. I shall uphold the Constitution and Bylaws of the AMA and the South Dakota State Medical Association. I shall champion the cause of freedom in medical practice and freedom for all my fellow Americans.

I do solemnly swear that I will discharge the duties of this office to be the best of my ability, so help me God.

DISTINGUISHED SERVICE AWARD

- Started in 1951—T. F. Riggs, M.D., Pierre (deceased)
1952—H. Russell Brown, M.D., Watertown (deceased)
1953—Guy Van Demark, M.D., Sioux Falls (deceased)
1954—J. C. Ohlmacher, M.D., Vermillion (deceased)
1955—R. G. Mayer, M.D., Aberdeen (deceased)
1956—J. C. Ohlmacher, M.D., Vermillion (deceased)
1957—W. E. Donahoe, M.D., Sioux Falls (deceased)
1958—Drs. J. C. Hagin (deceased), M. W. Pangburn (deceased), and James DeGeest, Miller
1958—J. F. Brenckle, M.D., Superior, Wisc. (deceased)
1958—Mrs. Agnes Holdridge, Madison
1959—Walter L. Hard, Ph.D., Vermillion
1959—Rev. and Mrs. Robert O. Bates, Sturgis
1959—R. M. Kilgard, M.D., Watertown (deceased)
1960—L. J. Pankow, M.D., Sioux Falls (deceased)
1961—Gregg M. Evans, Ph.D., Custer
1962—Edward Shaw, Ph.D., Vermillion (deceased)
1963—Arthur A. Lampert, M.D., Rapid City
1964—John C. Foster, Phoenix, Arizona
1965—A. P. Reding, M.D., Marion
1966—Mrs. C. Rodney Stoltz, Watertown
1967—Mrs. William Fish, Watertown

- 1968—G. J. Bloemendaal, M.D., Ipswich
1969—F. W. Haas, M.D., Yankton (deceased)
1970—Paul Bunker, M.D., Aberdeen (deceased)
1971—E. T. Lietzke, M.D., Beresford (deceased)
1972—C. B. McVay, M.D., Yankton
1973—G. E. Tracy, M.D., Watertown
1974—J. A. Muggly, M.D., Madison
1975—Harvey Wollman, Hitchcock
1976—R. H. Quinn, M.D., Sioux Falls
1977—E. H. Heinrichs, M.D., Vermillion
1978—John Olson, Rapid City, and Evans Nord, Sioux Falls
1979—Helen Jane Hare, M.D., Rapid City
1980—Warren Jones, M.D., Sioux Falls
1981—Saul Friefeld, M.D., Brookings

COMMUNITY SERVICE AWARD

- 1961—R. A. Buchanan, M.D., Huron (deceased)
1962—Roland F. Hubner, M.D., Yankton
1963—George W. Mills, M.D., Wall (deceased)
1964—John C. Hagin, M.D., Miller (deceased)
1965—Alonzo P. Peeke, M.D., Volga
1966—Hugo C. Andre, M.D., Vermillion (deceased)
1967—G. Robert Bartron, M.D., Watertown
1968—M. M. Morrissey, M.D., Pierre (deceased)
1969—N. J. Sundet, M.D., Kadoka (deceased)
1970—W. H. Saxton, M.D., Huron (deceased)
1971—R. E. Van Demark, M.D., Sioux Falls
1972—R. H. Hayes, M.D., Wall
1973—B. F. King, M.D., Aberdeen (deceased)
1974—M. C. Tank, M.D., Brookings
1975—Karl Wegner, M.D., Sioux Falls
1976—John T. Elston, M.D., Rapid City
1977—W. F. Stanage, M.D., Yankton
1978—C. S. Roberts, Jr., M.D., Brookings
1979—C. J. McDonald, M.D., Sioux Falls
1980—E. A. Johnson, M.D., Milbank
1981—J. A. Muggly, M.D., Madison

AESCULAPIUS AWARD

- 1966—Paul R. Leon, M.D.
Walter Miller, M.D., Aberdeen
1968—H. Phil Gross, M.D., Sioux Falls

FIFTY YEAR CLUB MEMBERS

- C. V. Auld, Plankinton (deceased)
G. J. Bloemendaal, M.D., Ipswich
W. C. Brinkman, M.D., Sisseton
R. A. Buchanan, M.D., Huron (deceased)
John L. Calene, M.D., California (deceased)
Myrtle Carney, M.D., Ft. Worth, Texas
J. C. Clark, M.D., Sioux Falls (deceased)
F. L. Class, M.D., Huron (deceased)
M. E. Cogswell, M.D., Wolsey (deceased)
J. Cook, M.D., Bonesteel (deceased)
Harold L. Crane, M.D., Avon, Conn. (deceased)
S. A. Donahoe, M.D., Sioux Falls (deceased)

W. E. Donahoe, M.D., Sioux Falls (deceased)
 V. W. Embree, M.D., Pierre (deceased)
 W. D. Farrell, M.D., Aberdeen (deceased)
 R. B. Fleeger, M.D., Lead (deceased)
 R. R. Fisk, M.D., Flandreau (deceased)
 F. W. Freyberg, M.D., Mitchell
 E. E. Gage, M.D., Sioux Falls (deceased)
 D. A. Gregory, M.D., Glasgow, Mont.
 E. H. Grove, M.D., Arlington (deceased)
 J. C. Hagin, M.D., Miller (deceased)
 Lyle Hare, M.D., Spearfish (deceased)
 John F. Hill, M.D., Yankton
 J. A. Hohf, M.D., Yankton (deceased)
 F. S. Howe, M.D., Deadwood (deceased)
 A. H. Hovne, M.D., Salem (deceased)
 A. S. Jackson, M.D., Rapid City (deceased)
 R. J. Jackson, M.D., Hot Springs (deceased)
 J. A. Jacotel, M.D., Milbank (deceased)
 G. T. Jordan, M.D., Vermillion (deceased)
 F. F. Keene, M.D., Wessington Springs (deceased)
 Ray Lemley, M.D., Rapid City
 J. H. Lloyd, M.D., Mitchell
 O. J. Mabee, M.D., Mitchell
 P. V. McCarthy, M.D., Aberdeen (deceased)
 G. W. Mills, M.D., Wall (deceased)
 B. C. Murdy, M.D., Aberdeen (deceased)
 T. F. O'Toole, M.D., Rapid City (deceased)
 N. T. Owen, M.D., Rapid City (deceased)
 L. L. Parke, M.D., Canton (deceased)
 A. P. Peeke, M.D., Volga
 M. O. Pemberton, M.D., Deadwood (deceased)
 R. J. Quinn, M.D., Sioux Falls (deceased)
 F. J. Radusch, M.D., California
 T. B. Ranney, M.D., Aberdeen (deceased)
 T. F. Riggs, M.D., Pierre (deceased)
 I. R. Salladay, M.D., Ft. Meade (deceased)
 W. H. Saxton, M.D., Huron (deceased)
 H. L. Saylor, M.D., Huron (deceased)
 C. E. Sherwood, M.D., Brookings (deceased)
 Myron Tank, M.D., Brookings
 F. J. Tobin, M.D., Mitchell (deceased)
 Leonard W. Tobin, M.D., Mitchell
 J. S. Tschetter, M.D., Huron (deceased)
 F. W. Valkenaar, M.D., Chancellor (deceased)
 G. E. Van Demark, M.D., Sioux Falls (deceased)
 H. P. Volin, M.D., Lennox (deceased)
 C. H. Weishaar, M.D., Aberdeen (deceased)
 J. R. Westaby, M.D., Madison (deceased)
 G. E. Zimmerman, M.D., Missoula, Montana
 (deceased)

C. B. ALFORD AWARD

1974—Roscoe Dean, M.D., Wessington Springs
 1975—Gerald Tracy, M.D., Watertown
 1976—Robert Westaby, M.D., Hot Springs
 1977—Robert VanDemark, M.D., Sioux Falls
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Toward a Middle Ground in the Technology Debate in Obstetric Care

John P. Geyman, MD

In the last ten years there has been an intensifying debate over the extent and manner in which technology should be applied in the everyday care of obstetric patients. As a result of a concerted effort to improve the experience of the United States in perinatal mortality and morbidity, together with the development of new technologies to more closely monitor the course of pregnancy and labor, patterns of obstetric care have changed greatly across the country. Many hospitals, for example, require fetal monitoring for all patients in labor, and some proponents of fetal monitoring even assert that it is malpractice today to deny patients this service. In contrast to the advocates of high technology obstetrics, whose principal concerns in obstetric care involve the prevention and treatment of **disease**, is a growing group of advocates for individualized use of technology based on the predominant normalcy of pregnancy, labor, and delivery as a **natural** process. Arguments in favor of limited and selective use of technology in obstetric care have included concerns over cost, effectiveness, and risks of technologic interventions, and have stressed the importance of family centered obstetrics with the active participation of women and couples in the choice of their obstetric care.

The cesarean section rate is a good indicator of the extent to which patterns of obstetric care have changed during the last ten years. The average cesarean section rate in this country tripled between 1970 and 1978 from 5.5 to 15.2 percent of deliveries. There is considerable regional variation in these figures with the highest cesarean section rates in the Northeast (17.6 percent in 1978). Although controversial, many studies indicate that fetal monitoring has played a major role in this increase, which has led to an additional direct cost of at least \$1200 to \$1500 per cesarean section and extension of the average hospital stay for obstetric delivery from about three days to one week.¹

In an effort to disentangle an array of conflicting studies and views on this important subject, a Consensus Development Conference sponsored by the National Institute of Child Health and Human Development was held in 1979. After intensive study and discussion of these issues involving input from a wide range of disciplines (obstetrics, pediatrics, epidemiology, ethics, law, sociology, and economics), the following conclusions were drawn.²

1. Fetal distress in labor cannot be assessed by considering a single parameter such as intermittent or continuous fetal heart rate. Because fetal heart rate patterns suggestive of hypoxia may occur in the absence of fetal distress, intermittent and continuous fetal heart rate assessment are screening, rather than diagnostic, techniques. Failure to appreciate this limitation may lead to inappropriate clinical decisions.

2. The weight of present evidence from prospective and retrospective analyses shows no apparent effect of electronic fetal monitoring upon perinatal mortality and morbidity in low-risk patients. As maternal and fetal risk increases, there is a trend suggesting a beneficial effect of electronic fetal monitoring upon intrapartum and neonatal morbidity and mortality. Specific obstetric risk factors especially amenable to intervention via electronic fetal monitoring have not been completely enumerated.

3. Periodic auscultation of the fetal heart rate (for 30 seconds every 15 minutes in the first stage of labor and every 5 minutes during the second stage, immediately following a contraction) is an acceptable method of assessment of fetal condition for women at low risk of intrapartum fetal distress.

4. The use of electronic fetal monitoring should be strongly considered in high risk patients. Some of the high risk situations may include the following: (1) low birth weight, prematurity, postmaturity, and intrauterine growth retardation, (2) medical complications of pregnancy, (3) meconium staining of the amniotic fluid, (4) intrapartum obstetrical complications, (5) the use of oxytocin in labor, and (6) the presence of abnormal auscultatory findings.

5. Further studies of the variables affecting the relationship between electronic monitoring and cesarean section and its complications are necessary and should be encouraged.

Two papers in this issue of this journal examine patterns of obstetric care from two particular perspectives. Brody and Thompson argue that much of currently accepted obstetric practice is based upon a maximin strategy (ie, that approach that makes the best of the worst possible outcome without regard to the expected frequency of that outcome). They present and assess the evidence for and against the widespread use of various obstetric interventions, and explore approaches to individualize obstetric care based upon reasonable risk assessment and the documented effectiveness of specific interventions.³ McClain examines the perspectives and attitudes of the patient with respect to the influences that bear upon women's choice of birth in hospitals or at home.⁴ Of special interest are the ways in which women's perceptions of medical risk differ from today's mainstream doctrine of obstetric care held by physicians.

As more graduates emerge from family practice residency programs, a steadily increasing number of family physicians will provide obstetric care in this country. The family physician is ideally situated to provide family centered obstetric care based upon individualized risk assessment and selective use of obstetric interventions, utilizing consultation as needed. A shift of the technology debate in obstetrics toward a more reasonable middle ground is both needed and in the public interest.

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4. McClain C: Women's choice of home or hospital birth. *J Fam Pract* 12:1033, 1981



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BRIEF SUMMARY

INDICATIONS AND USAGE

For the prevention and treatment of nocturnal recumbency leg muscle cramps

CONTRAINDICATIONS

Quinamm may cause fetal harm when administered to a pregnant woman. Congenital malformations in the human have been reported with the use of quinine, primarily with large doses (up to 30 g) for attempted abortion. In about half of these reports the malformation was deafness related to auditory nerve hypoplasia. Among the other abnormalities reported were limb anomalies, visceral defects, and visual changes. In animal tests, teratogenic effects were found in rabbits and guinea pigs and were absent in mice, rats, dogs, and monkeys. Quinamm is contraindicated in women who are or may become pregnant. If this drug is used during pregnancy, or if the patient becomes pregnant while taking this drug, the patient should be apprised of the potential hazard to the fetus.

Because of the quinine content, Quinamm is contraindicated in patients with known quinine hypersensitivity and in patients with glucose-6-phosphate dehydrogenase (G-6-PD) deficiency.

Since thrombocytopenic purpura may follow the administration of quinine in highly sensitive patients, a history of this occurrence associated with previous quinine ingestion contraindicates its further use. Recovery usually occurs following withdrawal of the medication and appropriate therapy.

This drug should not be used in patients with tinnitus or optic neuritis or in patients with a history of blackwater fever.

WARNINGS

Repeated doses or overdosage of quinine in some individuals may precipitate a cluster of symptoms referred to as cinchonism. Such symptoms, in the mildest form, include ringing in the ears, headache, nausea, and slightly disturbed vision, however, when medication is continued or after large single doses, symptoms also involve the gastrointestinal tract, the nervous and cardiovascular systems, and the skin.

Hemolysis (with the potential for hemolytic anemia) has been associated with a G-6-PD deficiency in patients taking quinine. Quinamm should be stopped immediately if evidence of hemolysis appears.

If symptoms occur, drug should be discontinued and supportive measures instituted. In case of overdosage, see OVERDOSAGE section of prescribing information.

PRECAUTIONS

General

Quinamm should be discontinued if there is any evidence of hypersensitivity. (See CONTRAINDICATIONS.) Cutaneous flushing, pruritus, skin rashes, fever, gastric distress, dyspnea, ringing in the ears, and visual impairment are the usual expressions of hypersensitivity, particularly if only small doses of quinine

have been taken. Extreme flushing of the skin accompanied by intense, generalized pruritus is the most common form. Hemoglobinuria and asthma from quinine are rare types of idiosyncrasy.

In patients with atrial fibrillation, the administration of quinine requires the same precautions as those for quinidine. (See Drug Interactions.)

Drug Interactions

Increased plasma levels of digoxin and digitoxin have been demonstrated in individuals after concomitant quinidine administration. Because of possible similar effects from use of quinine, it is recommended that plasma levels for digoxin and digitoxin be determined for those individuals taking these drugs and Quinamm concomitantly.

Concurrent use of aluminum-containing antacids may delay or decrease absorption of quinine.

Cinchona alkaloids, including quinine, have the potential to depress the hepatic enzyme system that synthesizes the vitamin K-dependent factors. The resulting hypoprothrombinemic effect may enhance the action of warfarin and other oral anticoagulants.

The effects of neuromuscular blocking agents (particularly pancuronium, succinylcholine, and tubocurarine) may be potentiated with quinine, and result in respiratory difficulties.

Urinary alkalinizers (such as acetazolamide and sodium bicarbonate) may increase quinine blood levels with potential for toxicity.

Drug Laboratory Interactions

Quinine may produce an elevated value for urinary 17-ketogenic steroids when the Zimmerman method is used.

Carcinogenesis, Mutagenesis, Impairment of Fertility

A study of quinine sulfate administered in drinking water (0.1%) to rats for periods up to 20 months showed no evidence of neoplastic changes.

Mutation studies of quinine (dihydrochloride) in male and female mice gave negative results by the micronucleus test. Intraperitoneal injections (0.5 mM/kg) were given twice, 24 hours apart. Direct *Salmonella typhimurium* tests were negative, when mammalian liver homogenate was added, positive results were found.

No information relating to the effect of quinine upon fertility in animal or in man has been found.

Pregnancy

Category X. See CONTRAINDICATIONS.

Nonteratogenic Effects

Because quinine crosses the placenta in humans, the potential for fetal effects is present. Stillbirths in mothers taking quinine have been reported in which no obvious cause for the fetal deaths was shown. Quinine in toxic amounts has been associated with abortion. Whether this action is always due to direct effect on the uterus is questionable.

Nursing Mothers

Caution should be exercised when Quinamm is given to nursing women because quinine is excreted in breast milk (in small amounts).

ADVERSE REACTIONS

The following adverse reactions have been reported with Quinamm in therapeutic or excessive dosage. (Individual or multiple symptoms may represent cinchonism or hypersensitivity.)

Hematologic: acute hemolysis, thrombocytopenic purpura, agranulocytosis, hypoprothrombinemia.

CNS: visual disturbances, including blurred vision with scotomata, photophobia, diplopia, diminished visual fields, and disturbed color vision; tinnitus, deafness, and vertigo; headache, nausea, vomiting, fever, apprehension, restlessness, confusion, and syncope.

Dermatologic/allergic: cutaneous rashes (urticarial, the most frequent type of allergic reaction; papular or scarlatiniform), pruritus, flushing of the skin, sweating, occasional edema of the face.

Respiratory: asthmatic symptoms.

Cardiovascular: anginal symptoms.

Gastrointestinal: nausea and vomiting (may be CNS-related), epigastric pain.

DRUG ABUSE AND DEPENDENCE

Tolerance, abuse, or dependence with Quinamm has not been reported.

OVERDOSAGE

See prescribing information for a discussion on symptoms and treatment of overdose.

DOSSAGE AND ADMINISTRATION

1 tablet upon retiring. If needed, 2 tablets may be taken nightly—1 following the evening meal and 1 upon retiring.

After several consecutive nights in which recumbency leg cramps do not occur, Quinamm may be discontinued in order to determine whether continued therapy is needed.

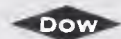
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75 mg controlled-release tablets

the #1 prescribed anorectic

An effective short-term adjunct in an indicated weight loss program

Overweight patients in certain diagnostic categories often require strict obesity control. Diethylpropion hydrochloride has been reported useful in obese patients with certain complications. While it is not suggested that Tenuate in any way reduces these complications in the overweight, it may have a useful place as a short-term adjunct in a prescribed dietary regimen. Tenuate should not be administered to patients with severe hypertension; see additional Precautions and Adverse Reactions on this page.

In uncomplicated obesity

Many patients, on the other hand, present with excess fat but no disease. While this condition is often termed uncomplicated obesity, complications of both a social and a psychologic nature may be distressingly real for the patients. In these cases, a short-term regimen of Tenuate can help reinforce your dietary counsel during the important early weeks of an indicated weight loss program.

Clinical effectiveness

The anorectic effectiveness of diethylpropion hydrochloride is well documented. No less than 18 separate double-blind, placebo-controlled studies attest to its usefulness in daily practice.¹ And the unique chemistry of Tenuate provides "...anorectic potency with minimal overt central nervous system or cardiovascular stimulation."² Compared with the amphetamines, diethylpropion has minimal potential for abuse.

**Tenuate—it makes sense.
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References: 1. Citations available on request from Merrell Dow Pharmaceuticals Inc., Cincinnati, Ohio 45215. 2. Hoekenga M. T. et al: A comprehensive review of diethylpropion hydrochloride. In *Central Mechanisms of Anorectic Drugs*, S. Garattini and R. Samanin, Ed., New York. Raven Press, 1978, pp. 391-404.

Tenuate[®] 
(diethylpropion hydrochloride USP)

Tenuate Dospan[®] 
(diethylpropion hydrochloride USP)
controlled-release

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.

CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. When central nervous system active agents are used, consideration must always be given to the possibility of adverse interactions with alcohol. **Drug Dependence:** Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. **Use in Pregnancy:** Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. **Use in Children:** Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: **Cardiovascular:** Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. **Central Nervous System:** Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. **Gastrointestinal:** Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. **Allergic:** Urticaria, rash, ecchymosis, erythema. **Endocrine:** Impotence, changes in libido, gynecomastia, menstrual upset. **Hematopoietic System:** Bone marrow depression, agranulocytosis, leukopenia. **Miscellaneous:** A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in mid-evening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in mid-morning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine[®]) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of June, 1980

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SD

President's Page



I have just returned from the 1981 Annual Meeting of the AMA. I was impressed! The AMA House of Delegates made decisions endorsing a new functional profile for the AMA, plus decisions in the areas of medical education, scientific medicine, and governmental control of medicine. In fact every important concern of American medicine was discussed in at least one reference committee and everyone had the opportunity to speak to the issues. I was impressed with the magnitude of the meeting. It is amazing how much work is accomplished in just a few days. I hope you have read **American Medical News** of June 26 reviewing the results of the meeting. It was a superb meeting!

The AMA needs new members to strengthen its voice as it represents you and me. Seventy-five percent of SDSMA members already belong to the AMA. However, I feel if the remaining 25% of you who are not AMA members could attend one meeting of the AMA House of Delegates, you would all join right away!

AMA dues are going to be \$285 in 1982—a \$35 increase. This is the first dues increase since 1976. There are reduced dues for first and second year members, military physicians, medical students, residents and retired or disabled physicians. It's a bargain when you consider what the AMA does for you.

Just ask yourself these questions—

What is it worth to have a national association that has represented each of you so successfully through several decades in preserving a pluralistic

health care system—one that preserves freedom of choice for physicians and patients alike?

What is it worth to have a national association that during the past few years has been so successful in representing you in the Congress and in the courts?

What is it worth to have a strong national political arm, AMPAC, who supports, in a bipartisan way, those congressional candidates who are committed to the best ends, not only of medicine and health, but of government itself?

What is it worth to have a national organization ready to adjust to any changes affecting your freedom to provide your patients the best of medical care?

What is it worth to have a national organization willing to take a careful introspective look at itself and to reorder its functions, activities and resources to better represent the AMA membership?

Organized medicine's primary purpose is to help you in your efforts to provide quality care for your patients. It is important for you to participate—be active in your district medical society, be active in SDSMA activities and **join** the AMA.

TOGETHER—we can make a difference!

Sincerely yours,

Bruce Lushbough, M.D., President
South Dakota State Medical Association

South Dakota State Medical Association Roster—1981

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Delaney, W. A., Jr.	Mitchell	*Lloyd, J. H.	Mitchell	Skogmo, B. R.	Mitchell
Dilger, J.	Mitchell	Mabee, J. O.	Mitchell	*Tobin, L. W.	Mitchell
Gere, R. G.	Mitchell	*Mabee, O. J.	Mitchell	Visani, S.	Mitchell
Gillis, F. D.	Mitchell	Margallo, L.	Mitchell	*Vonburg, V. R.	Mitchell
Haley, M.	Mitchell	McCann, J. P.	Parkston	Vose, J. L.	Mitchell
Hansen, R. J.	Kimball	Moller, C.	Mitchell	Weatherill, D. W.	Mitchell
Hermann, H. T.	Mitchell	Monson, C. D.	Parkston	*Weber, R. A.	Mitchell
Heth, S.	Mitchell	Mueller, E. H.	Tripp	Williams, H. S.	Mitchell

SIOUX FALLS DISTRICT No. 7

Pres., L. W. Finney, M.D. Sec., Gail Benson, M.D. Treas., R. C. Johnson, M.D.

Abu-Ghazaleh, S.	Sioux Falls	Church, W. G.	Sioux Falls	Greenfield, D. L.	Sioux Falls
*Alcorn, F. A.	Sioux Falls	Clark, E. T.	Sioux Falls	Gregg, J. B.	Sioux Falls
Alvine, F. G.	Sioux Falls	Cloar, R.	Sioux Falls	Groote, C.	Sioux Falls
Amundson, Loren	Sioux Falls	*Cottam, G. I. W.	Sioux Falls	Gross, H. Phil	Sioux Falls
Anderson, C. W.	Sioux Falls	Cutshall, V. H.	Sioux Falls	*Grove, M.S.	Sioux Falls
Anderson, Edward	Sioux Falls	Cutshall, V. K.	Sioux Falls	Gunnarson, R. E.	Sioux Falls
Anderson, T. R.	Sioux Falls	Daw, E. F.	Sioux Falls	Gutch, C. F.	Sioux Falls
Anderson, W. R.	Sioux Falls	DeClark, R. P.	Sioux Falls	Gutnik, L.	Sioux Falls
Angelos, T.	Canton	Devick, J. S.	Colton	Hartzell, A.	Sioux Falls
Arneson, W. A.	Sioux Falls	Donahoe, J. W.	Sioux Falls	Henriekson, L.	Sioux Falls
Aspaas, P. K.	Dell Rapids	Drymalski, W.	Sioux Falls	Henriekson, R.	Sioux Falls
Aspaas, Paul Jr.	Sioux Falls	Dzintars, V.	Sioux Falls	Hermanson, J. M.	Valley Springs
Assam, Sam	Sioux Falls	Easton, J.	Sioux Falls	Holt, B.	Sioux Falls
Barker, J. D.	Sioux Falls	*Eirinberg, I.	Sioux Falls	Hosen, R. S.	Sioux Falls
Barlow, J. F.	Sioux Falls	Elkjer, N.	Sioux Falls	Hoskins, John	Sioux Falls
Barnett, G. L.	Sioux Falls	Elson, D.	Sioux Falls	Hoxtell, Eugene	Sioux Falls
Belatti, R. G.	Sioux Falls	English, G.	Sioux Falls	Humphreys, D.	Sioux Falls
Benson, G.	Sioux Falls	Ensborg, D.	Sioux Falls	Hurley, Brian	Sioux Falls
Bhatara, V.	Sioux Falls	Entwistle, F. R.	Sioux Falls	Hussain, Rifat	Sioux Falls
Bhatti, T. H.	Sioux Falls	Epp, D.	Freeman	Hyland, L.	Sioux Falls
Billion, J. J.	Sioux Falls	Farrell, H. W.	Sioux Falls	Ingvaldstad, J.	Sioux Falls
Billion, T. J., Jr.	Sioux Falls	Felker, James	Sioux Falls	Janis, J. B.	Sioux Falls
Blake, J.	Sioux Falls	Ferrell, M. R.	Sioux Falls	Jaqua, R. A.	Sioux Falls
Boade, W. A.	Sioux Falls	Finney, L. W.	Sioux Falls	Johnson, D. L.	Sioux Falls
*Breit, D. H.	Sioux Falls	*Fisk, R. G.	Dell Rapids	Johnson, P. S.	Sioux Falls
Brziea, S. M.	Sioux Falls	Flora, G. C.	Sioux Falls	Johnson, R. C.	Sioux Falls
Buey, C.	Beresford	Freeman, J. W.	Sioux Falls	Jones, W. L.	Sioux Falls
Burkhart, T.	Sioux Falls	Friess, R. W.	Sioux Falls	Kaufman, I. I.	Freeman
Burns, E. A.	Sioux Falls	Frost, D. M.	Sioux Falls	Kemp, E.	Sioux Falls
Burns, K.	Sioux Falls	Fuller, Wm. C.	Sioux Falls	Kennelly, Daniel	Sioux Falls
*Carney, M.	Texas	Gehring, S.	Sioux Falls	*King, L. M.	Sioux Falls
Carpenter, P.	Sioux Falls	Giebink, R. R.	Sioux Falls	Kittleson, H. O.	Sioux Falls
Carter, G.	Sioux Falls	Graham, Donald	Sioux Falls	Knowles, R. C.	Sioux Falls
Chalmers, J. H.	Sioux Falls	Grau, T.	Sioux Falls	Knudson, D.	Sioux Falls

Knutson, Dennis	Sioux Falls	Oakland, J.	Sioux Falls	Schultz, R. D.	Sioux Falls
*Kohlmeyer, F. C.	Sioux Falls	O'Brien, C.	Sioux Falls	Simmons, J. L.	Sioux Falls
Koob, K.	Sioux Falls	O'Brien, P.	Sioux Falls	Sittner, Larry	Sioux Falls
Lakstigala, P.	Sioux Falls	Ochsner, J. A.	Sioux Falls	Slattery, M.	Sioux Falls
Lang, Durward	Sioux Falls	*Ogborn, R. J.	Sioux Falls	*Smith, G.	Sioux Falls
Langdon, J.	Sioux Falls	Opheim, W. L.	Sioux Falls	Solberg, L.	Sioux Falls
Larson, Leland J.	Sioux Falls	Opheim, W. O. V.	Sioux Falls	*Stahmann, F.	Sioux Falls
Leander, R. B.	Sioux Falls	Orr, R. T.	Sioux Falls	Stassen, M.	Sioux Falls
Lee, S. G.	Sioux Falls	Ortmeier, Denny	Sioux Falls	*Steiner, P. K.	California
Looby, T.	Sioux Falls	Owens, L.	Sioux Falls	Stensland, V.	Sioux Falls
Loos, G. D.	Sioux Falls	Parry, R.	Sioux Falls	Stoltz, C. R.	Sioux Falls
Lovrien, F.	Sioux Falls	Pasek, E. A.	Sioux Falls	Swanson, P.	Sioux Falls
Madison, Dean	Sioux Falls	Patterson, H.	Lennox	Talley, Robert	Sioux Falls
Magnuson, G.	Sioux Falls	Payne, H.	Sioux Falls	Tam, Guy	Sioux Falls
*Maresh, E. R.	Sioux Falls	Pekas, M.	Sioux Falls	Thatcher, L. G.	Sioux Falls
Marschke, R.	Sioux Falls	Petereit, M. F.	Sioux Falls	Tschetter, L. K.	Sioux Falls
Marvel, J.	Sioux Falls	Peters, E. H.	Sioux Falls	Tschetter, R. T.	Sioux Falls
Mark, C.	Viborg	Petres, A.	Salem	Ulus, M.	Sioux Falls
McDonald, C. J.	Sioux Falls	Pitt-Hart, Barry T.	Sioux Falls	Van Demark, R. E.	Sioux Falls
*McGreevy, John	Sioux Falls	Putnam, W.	Sioux Falls	Vander Wonde, L.	Sioux Falls
McGreevy, E. J.	Sioux Falls	Quale, J.	Sioux Falls	Villa, Jose	Freeman
McGreevy, P. S.	Sioux Falls	Quinn, R. H.	Sioux Falls	Vogt, H. B.	Sioux Falls
McHardy, B. R.	Aurora	Randall, B.	Sioux Falls	Volin, V. V.	Sioux Falls
McMillin, J.	Sioux Falls	Raszkowski, R.	Sioux Falls	Wagner, L.	Sioux Falls
Morris, A. D.	Sioux Falls	Read, R.	Sioux Falls	Waltner, Lonnie	Bridgewater
Munson, D.	Sioux Falls	Regier, E.	Canton	Walton, J. E.	Sioux Falls
Mutch, M. G.	Sioux Falls	Reynolds, James	Sioux Falls	Watson, Wm.	Sioux Falls
Naughton, G.	Sioux Falls	Richards, George	Sioux Falls	Wegner, K. H.	Sioux Falls
Nelson, Earl	Viborg	Rossing, D.	Sioux Falls	White, T. C.	Sioux Falls
Nelson, R. A.	Sioux Falls	Rossing, W. O.	Sioux Falls	Wierda, D. R.	Sioux Falls
Nelson, R. E.	Sioux Falls	Rost, M.	Sioux Falls	Williams, B. J.	Sioux Falls
Nice, Richard	Sioux Falls	Rutt, Carl	Sioux Falls	Willix, Robert	Sioux Falls
Nielsen, J.	Dell Rapids	Salmela, S.	Sioux Falls	Wilson, T. M.	Sioux Falls
Noll, S.	Minn.	Sanchez, G.	Sioux Falls	Wyatt, George	Sioux Falls
Nordstrom, D.	Sioux Falls	Sanderson, E. W.	Sioux Falls	Wyatt, R.	Sioux Falls

YANKTON
DISTRICT No. 8

Pres., H. J. Fletcher, M.D. Sec., Thomas Olson, M.D. Treas., Roger Nutt, M.D.

Bean, David	Yankton	Johnson, B.	Vermillion	Reding, A. P.	Marion
Brinkman, R.	Yankton	Johnson, T. C.	Yankton	Saloom, H.	Tyndall
Brookman, B. T.	Wagner	Johnson, V.	Vermillion	Saoi, N. B.	Yankton
Dendinger, Wm.	Vermillion	Kalda, E. F.	Platte	Sattler, T. H.	Yankton
Fletcher, H.	Vermillion	Lyso, M.	Yankton	*Sebring, F. U.	Vermillion
Flom, J.	Yankton	McVay, C. B.	Yankton	Smith, D.	Yankton
Foley, R. J.	Tyndall	Messner, F.	Yankton	Stanage, W. F.	Yankton
Halverson, K.	Yankton	Neumayr, R. J.	Yankton	Steele, J. P.	Yankton
Heinrichs, E. H.	Vermillion	Nutt, R.	Yankton	Stephenson, D.	Yankton
Held, G.	Yankton	Olson, Thomas	Vermillion	Sternquist, J.	Yankton
*Hill, J. F.	Yankton	Pascale, C. C.	Centerville	Stevens, J.	Yankton
Hollerman, Chas.	Vermillion	Petersen, L.	Yankton	Thompson, R. F.	Yankton
Holzwarth, D. R.	Yankton	Porter, Richard I.	Yankton	Thornton, R. R.	Yankton
Honke, R. W.	Wagner	Price, Ronald	Armour	Tidd, J. T.	Yankton
Hubner, J.	Yankton	Pullen, M.	Yankton	Tuan, C.	Yankton
*Hubner, R. F.	Yankton	Quick, Wm.	Yankton	Turner, C. R.	Vermillion
Isburg, Carroll	Yankton	Radack, Morris	Yankton	Willcockson, John	Yankton
Jacobsen, J. J.	Yankton	Ranney, B.	Yankton	Willcockson, T. H.	Yankton
Jameson, G. M.	Yankton	Reaney, D. B.	Yankton		

BLACK HILLS
DISTRICT No. 9

Pres., N. R. Whitney, M.D. Sec., A. J. Barrett, M.D.

Ahrlin, H. L.	Rapid City	Bailey, J. D.	Rapid City	Berkebile, Dale	Rapid City
Ahrlin, H. L., Jr.	Rapid City	Bareis, R. J.	Rapid City	Berry, D.	Sturgis
Allen, Bruce	Rapid City	Barrett, A. J.	Rapid City	Bloemendaal, R. D.	Rapid City
Anderson, A. B.	Lead	Bedingfield, J. R.	Rapid City	Blunck, C. J.	Rapid City
Arnold, G.	Rapid City	Behrens, C. L.	Rapid City	*Borgmeyer, H. J.	Rapid City
Authier, N.	Rapid City	Bergeron, Dale	Rapid City	Boyce, R. A.	Rapid City

Boyer, D. W. Rapid City
 Branch, Robert Rapid City
 *Bray, R. B. Rapid City
 Brown, Michael Spearfish
 Burnap, D. W. Rapid City
 Burnett, R. Rapid City
 Butz, Gerald Rapid City
 Carlson, G. Rapid City
 Carson, L. E. Arkansas
 *Clark, B. S. Spearfish
 Cline, J. A. North Carolina
 Cornford, R. C. Rapid City
 Dewald, A. Rapid City
 Drummond, R. Rapid City
 Dzintars, P. F. Rapid City
 Ebbert, Larry Rapid City
 Elston, J. T. Rapid City
 Engel, C. Rapid City
 Ferrell, R. Rapid City
 Finley, R. C. Rapid City
 Forshner, R. Hot Springs
 Freimark, L. G. Rapid City
 Fromm, H. E. Rapid City
 Frost, H. L. Rapid City
 Gilbert, F. J. Ft. Meade
 Golliher, W. N. Spearfish
 Gwinn, C. B. Rapid City
 Haas, S. Rapid City
 Hafner, Daniel Rapid City
 Hamm, Joseph Sturgis
 Hare, H. J. Rapid City
 Harris, R. H. Rapid City
 Haugan, H. O. Rapid City
 Heideprcim, G. Rapid City
 Henry, Thomas Rapid City
 Herbrandson, C. R. Spearfish
 Hercules, C. Rapid City
 Hewitt, J. M. Rapid City

Howard, Wm. J. Rapid City
 Jackson, J. Rapid City
 Jacobson, T. R. Hot Springs
 James, E. Rapid City
 Javurek, A. J. Vermillion
 Jenter, G. W. Sturgis
 Jerde, O. M. Rapid City
 Johnson, Robert K. Rapid City
 Jones, W. E. Sturgis
 *Kegaries, D. L. Arizona
 Kelley, D. H. Rapid City
 Kelts, K. A. Rapid City
 Klar, W. Fort Meade
 Knecht, J. Martin
 Kovarik, J. A. Rapid City
 Kovarik, R. A. Rapid City
 Kovarik, W. J. Rapid City
 Krafka, T. Rapid City
 Kullbom, J. Rapid City
 Kunz, J. A. Rapid City
 Kwan, F. P. Rapid City
 *Lampert, A. A. Rapid City
 *Lemley, R. E. Rapid City
 Liedtke, C. Sturgis
 Loos, C. Rapid City
 Lopez, A. Hot Springs
 Lucas, C. Hot Springs
 Mangulis, G. J. Philip
 Massa, L. L. Sturgis
 Mattson, W. Rapid City
 McGuigan, P. M. Rapid City
 *Mead, T. Spearfish
 *Merryman, M. P. Rapid City
 Meyers, W. Hot Springs
 Millea, R. P. Rapid City
 Mortimer, S. Rapid City
 Munson, H. B. Rapid City
 Neu, Norman Rapid City

Nord, A. Rapid City
 O'Sullivan, J. Belle Fourche
 Owen, G. S. Rapid City
 Palmerton, E. S. Rapid City
 Pavlovich, J. Wyoming
 Perry, Wm. J. Ft. Meade
 Pieczka, Jan Hot Springs
 *Radusch, F. J. California
 Reinoehl, W. Custer
 Renka, R. Rapid City
 Rosario, E. Rapid City
 Ruud, E. T. Hot Springs
 Sandvik, D. E. Rapid City
 Sanmartin, J. Rapid City
 *Saxton, A. J. Arizona
 Sejvar, J. P. Rapid City
 Shining, H. S. Rapid City
 Slingsby, J. B. Rapid City
 Strand, R. D. Rapid City
 Sutliff, W. Rapid City
 Swisher, L. P. Kadoka
 Tesar, C. E. Rapid City
 Theissen, H. H. Rapid City
 Tracer, C. L. Rapid City
 Trinidad, R. Deadwood
 Tschetter, W. R. Rapid City
 Van Etten, D. Rapid City
 Voge, Kenneth Rapid City
 Welsh, Gary Rapid City
 Westaby, R. S. Hot Springs
 Whitney, N. R. Rapid City
 Wicks, Dennis Custer
 *Williams, F. R. Rapid City
 Wood, G. F. Rapid City
 Wright, Paul Rapid City
 Yackley, J. V. Rapid City
 Yamada, A. Rapid City
 Zanka, J. A. Rapid City

ROSEBUD DISTRICT No. 10

Pres., John Malm, M.D. Sec., Robert Stiehl, M.D.

Bailey, Donald O'Neill, Neb.
 Hogrete, L. Gregory
 Malm, J. Gregory

Nemer, R. G. Gregory
 Stiehl, R. Winner

Sweet, E. P. Burke
 Thompson, M. George Gregory

NORTHWEST DISTRICT No. 11

Pres., James Collins, M.D. Sec., L. M. Linde, M.D.

Benson, M. C. Iowa
 Collins, J. D. Mobridge
 Henderson, B. J. Mobridge
 Johnson, C. A. Lemmon

Linde, Leonard Mobridge
 *Nolan, B. P. Mobridge
 O'Connor, N. Eagle Butte
 Peterson, Jeffrey Mobridge

*Spiry, A. W. Mobridge
 Van Balen, C. Hoven
 Wunder, J. Mobridge
 Yecha, David Gettysburg

WHETSTONE VALLEY DISTRICT No. 12

Pres., Joseph Kass, M.D. Sec., David Oey, M.D.

Bell, Eldon Webster
 *Brinkman, W. C. Sisseton
 Bucntipo, B. Texas
 *Gregory, D. A. Montana

Janavs, V. Milbank
 Johnson, E. A. Milbank
 Kass, Joseph Rosholt
 Mendoza, V. Sisseton

Nelson, L. F. Webster
 Oey, D. Sisseton
 Staub, D. W. Sisseton
 Vogelgesang, L. C. Webster

*—Indicates Honorary Membership

South Dakota State Medical Association Roster—1981

Membership—Alphabetical Listing

Abu-Ghazaleh, S.	Sioux Falls	Branch, R.	Rapid City	Eckrich, J. A., Jr.	Aberdeen
Adajar, A.	Bowdle	*Bray, R. B.	Rapid City	*Eirinberg, I.	Sioux Falls
Adams, H. P.	Huron	*Breit, D. H.	Sioux Falls	Elkjer, N.	Sioux Falls
Ahrlin, H. L.	Rapid City	Brinkman, R.	Yankton	Elson, D.	Sioux Falls
Ahrlin, H. L., Jr.	Rapid City	*Brinkman, W. C.	Sisseton	Elston, J. T.	Rapid City
Albano, P.	Aberdeen	Broadhurst, K. A.	Aberdeen	Engel, C.	Rapid City
*Alcorn, F. A.	Sioux Falls	Brookman, B. T.	Wagner	English, G.	Sioux Falls
Allen, Bruce	Rapid City	Brown, M.	Spearfish	Ensberg, D. L.	Sioux Falls
Allen, S. W.	Watertown	Brown, R.	Aberdeen	Entwistle, F. R.	Sioux Falls
Altman, S.	Aberdeen	Brzica, S. M.	Sioux Falls	Epp, D. L.	Freeman
Alvine, F. G.	Sioux Falls	Buchanan, D.	Huron	Fahrenwald, M.	Aberdeen
Amundson, Loren	Sioux Falls	Bucy, Christine	Beresford	Farrell, H. W.	Sioux Falls
Anderson, A. B.	Lead	Buentipo, B.	Texas	Fedt, Donald	Watertown
Anderson, C. W.	Sioux Falls	Bunker, T.	Aberdeen	Felker, J.	Sioux Falls
Anderson, Edward	Sioux Falls	Burkhart, T.	Sioux Falls	Ferrell, M. R.	Sioux Falls
Anderson, J. A.	Madison	Burnap, D. W.	Rapid City	Ferrell, R.	Rapid City
Anderson, T. R.	Sioux Falls	Burnett, R.	Rapid City	Finley, R. C.	Rapid City
Anderson, W. R.	Sioux Falls	Burns, E. A.	Sioux Falls	Finney, L.	Sioux Falls
Angelos, T.	Canton	Burns, K. R.	Sioux Falls	*Fisk, R. G.	Flandreau
Appelwick, J.	Madison	Butz, Gerald	Rapid City	Fletcher, H.	Vermillion
Argabrite, J. W.	Watertown	Carlson, G.	Rapid City	Flom, J.	Yankton
Arneson, W. A.	Sioux Falls	*Carney, M.	Texas	Flora, G.	Sioux Falls
Arnold, G.	Rapid City	Carpenter, P.	Sioux Falls	Foley, R. J.	Tyndall
Askwig, L. C.	Pierre	Carson, L. E.	Arkansas	Forshner, R.	Hot Springs
Aspaas, P. K.	Dell Rapids	Carter, G.	Sioux Falls	*Fox, S. W.	California
Aspaas, Paul Jr.	Sioux Falls	Carter, P. B.	Aberdeen	Frazier, P.	Clear Lake
Assam, S.	Sioux Falls	Cavanaugh, D.	Huron	Freeman, J. W.	Sioux Falls
Authier, N.	Rapid City	Chalmers, J. H.	Sioux Falls	Freimark, L.	Rapid City
Baas, W.	Mitchell	Chang, J. P.	Aberdeen	Friefeld, S.	Minnesota
Bachmayer, Jay	Aberdeen	Chavier, Juan	Aberdeen	Friess, R. W.	Sioux Falls
Bailey, Don	O'Neill, Neb.	Christopher, John	Aberdeen	Fromm, H. E.	Rapid City
Bailey, J. D.	Rapid City	Chureh, Bill G.	Sioux Falls	Frost, D. M.	Sioux Falls
Bandiera, S.	Brookings	*Clark, B. S.	Spearfish	Frost, H. L.	Rapid City
Bareis, R. J.	Rapid City	Clark, C. J.	Watertown	Fuller, Wm. C.	Sioux Falls
Barker, J. D.	Sioux Falls	Clark, E. T.	Sioux Falls	Gehring, S.	Sioux Falls
Barlow, J. F.	Sioux Falls	Cline, J. A.	North Carolina	Gerber, B. C.	Aberdeen
Barnett, G. L.	Sioux Falls	Cloar, R.	Sioux Falls	Gere, R. G.	Mitchell
Barrett, A. J.	Rapid City	Collins, E. H.	Gettysburg	Giebink, R. R.	Sioux Falls
Bartholomew, K.	Faultkton	Collins, James	Mobridge	Gilbert, F. J.	Ft. Meade
Bartron, G. R.	Watertown	Cornford, R. C.	Rapid City	Gillis, F. D.	Mitchell
*Bartron, H. J., Jr.	Watertown	Cosand, M. R.	Pierre	Golliher, W. N.	Spearfish
Bean, David	Yankton	*Cottam, G. I. W.	Sioux Falls	Graham, D.	Sioux Falls
Bedingfield, J. R.	Rapid City	Cutshall, V. H.	Sioux Falls	Grau, T.	Sioux Falls
Behrens, C. L.	Rapid City	Cutshall, V. K.	Sioux Falls	Greenfield, D. L.	Sioux Falls
Belatti, R. G.	Sioux Falls			Gregg, J. B.	Sioux Falls
Bell, Eldon	Webster	Dappen, R.	Mitchell	*Gregory, D. A.	Montana
Bell, G. Robert	DeSmet	Davis, J.	Pierre	Groote, C.	Sioux Falls
Benson, G.	Sioux Falls	Daw, E. F.	Sioux Falls	Gross, H. Phil	Sioux Falls
Benson, M. C.	Iowa	Dean, Roscoe	Wess. Springs	*Grove, M. S.	Sioux Falls
Berg, S.	Redfield	Dean, Thomas	Wess. Springs	Gryte, C. F.	Huron
Bergeron, Dale	Rapid City	DeClark, R. P.	Sioux Falls	Guddal, W. N.	Watertown
Berkebile, D.	Rapid City	DeGeest, J. H.	Miller	Gunnarson, R. E.	Sioux Falls
Berry, D.	Sturgis	Delaney, R. J.	Mitchell	Gutch, C. F.	Sioux Falls
Berry, J. T.	Mitchell	Delaney, W. A., Jr.	Mitchell	Gutnik, L.	Sioux Falls
Betts, L. S.	Huron	Dendinger, Wm.	Vermillion	Gwinn, C. B.	Rapid City
Bhatara, V.	Sioux Falls	Desai, B. J.	Watertown	Haas, Stephen	Rapid City
Bhatti, T. H.	Sioux Falls	Devick, J. S.	Colton	Hafner, Daniel	Rapid City
Billion, J. J.	Sioux Falls	Dewald, A.	Rapid City	Haley, M.	Mitchell
Billion, T. J., Jr.	Sioux Falls	Dilger, J.	Mitchell	Halverson, K.	Yankton
Binder, C. F.	Chamberlain	Donahoe, J. W.	Sioux Falls	Hamm, Joseph	Sturgis
Blake, J.	Sioux Falls	Driver, I. E.	California	Hansen, R. J.	Kimball
*Bloemendaal, G. J.	Ipswich	Drummond, R.	Rapid City	Hanson, B.	Watertown
Bloemendaal, R. D.	Rapid City	Drymalski, W.	Sioux Falls	Hanson, W. O.	Huron
Blunek, C. F.	Rapid City	D'Souza, E. P.	Aberdeen	Hare, H. J.	Rapid City
Boade, W. A.	Sioux Falls	Dzintars, P. F.	Rapid City	Harlow, M. C.	Aberdeen
*Borgmeyer, H. J.	Rapid City	Dzintars, V.	Sioux Falls	Harris, Russell	Rapid City
Boyce, R. A.	Rapid City	Easton, J.	Sioux Falls	Hart, H.	Aberdeen
Boyer, D.	Rapid City	Ebbert, Larry	Rapid City	Hartzell, A.	Sioux Falls
Brakss, V.	Watertown	Eckrich, J. A.	Aberdeen	Haugan, H. O.	Rapid City

Hayes, R. H. Wall
Heidepreim, G. Rapid City
Heinrichs, E. Vermillion
Heisinger, R. Aberdeen
Held, G. Yankton
Henderson, B. Mobridge
Henrickson, L. Sioux Falls
Henrickson, R. Sioux Falls
Henry, Robert Brookings
Henry, T. Rapid City
Herbrandson, C. R. Spearfish
Hercules, C. Rapid City
Hermann, H. T. Mitchell
Hermanson, J. M. Valley Springs
Heth, S. Mitchell
Heupel, Alden R. Watertown
Hewitt, J. M. Rapid City
*Hill, J. F. Yankton
Hockett, R. D. Mitchell
Hofer, E. A. Huron
Hogrefe, L. Gregory
Hoffsten, P. Pierre
Hohm, Paul Huron
Hohm, R. Huron
Hohm, Theo Huron
Holland, L. W. Chamberlain
Hollerman, Chas. Vermillion
Holt, B. Sioux Falls
Holzwarth, D. R. Yankton
Honke, R. W. Wagner
Horne, J. Watertown
Hosen, R. S. Sioux Falls
Hoskins, John Sioux Falls
Hovland, James I. Aberdeen
Howard, Wm. J. Rapid City
Hoxtell, Eugene Sioux Falls
Huber, J. Redfield
Huber, T. Pierre
Hubner, J. Yankton
*Hubner, R. F. Yankton
Huet, G. M. Huron
Hughes, H. D. Clear Lake
Humphreys, D. Sioux Falls
*Huppler, E. G. Watertown
Hurley, B. Sioux Falls
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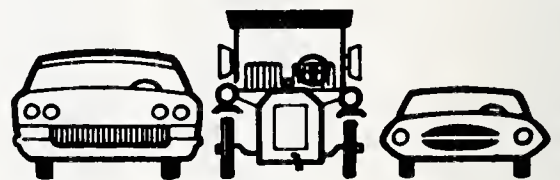
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Future Meetings

September

Advances in Clinical Nutrition, Sea Pines Resort, Hilton Head, SC, Sept. 3-4. Contact: Julie Bishop, A.S.P.E.N., Suite 810, 1025 Vermont Ave., N.W., Washington, D.C. 20005. Phone: (202) 638-5581.

Third Annual International Symposium on Clinical Otolaryngology, Radisson South Hotel, Minneapolis, MN, Sept. 3-5. Fee: \$350—Otolaryngologist and \$200—Audiologist. 21 hrs. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

AAMI 1981 Regional Meeting, Hyatt Regency, Chicago, IL, Sept. 9-10. Fee: \$90 for full day, \$45 for evening & half day. Contact: Renee Pietranglo, AAMI, Suite 602, 1901 N. Ft. Myer Dr., Arlington, VA 22209. Phone: (703) 525-4890.

The Third Biennial Leadbetter Symposium Pediatric Urology, West Bank Aud., Univ. of Minn., Minneapolis, MN, Sept. 9-12. Fee: \$350. 30 hrs. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Radiology/81 Gastrointestinal Radiology, West Bank Aud., U. of Minn., Minneapolis, MN, Sept. 14-18. Fee: \$350. 27 hrs. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Annual Autumn Seminar Obstetrics and Gynecology, Hyatt Regency, Minneapolis, MN, Sept. 16-18. Fee: \$230. 17 hrs. AAFP & AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

The Disabled Worker: Overcoming the System's Barriers, Radisson South Hotel, Bloomington, MN, Sept. 21-22. Fee: \$150. 14 hrs. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Third Adolescent Medicine and Health Care Conference, Focus on Adolescent Sexuality, Earle Brown Center, St. Paul Campus, St. Paul, MN, Sept. 23-24. Fee \$150—Physicians and \$80—Allied Health Prof. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone (612) 373-8012.

Fourth Annual Trauma Seminar, Hennepin County Medical Ctr., Minneapolis, MN, Sept. 24-26. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

October

Current Issues in Perinatal Care, Holiday Inn of the Northern Black Hills, Spearfish, SD, Oct. 12-13. 9.6 hrs. CME credits. Contact: Margo Varcoe, R.N. Prog. Dir., SDPA, 1100 S. Euclid Ave., Sioux Falls, SD 57105. Phone: (605) 339-6578.

AAMI 1981 Regional Meeting, L.A. Marriott, Los Angeles, CA, Oct. 12-13. Fee: \$90 for full day, \$45 for evening & half day. Contact: Renee Pietranglo, AAMI, Suite 602, 1901 N. Ft. Myer Dr., Arlington, VA 22209. Phone (703) 525-4890.

The 47th Annual Scientific Assembly of the American College of Chest Physicians, San Francisco Hilton Hotel & Civic Aud./Brooks Hall, San Francisco, CA, Oct. 25-29. 30 hrs. AMA Category I credits. Contact: Dept. of Education, AM. College of Chest Physicians, 911 Busse Highway, Park Ridge, IL 60068. Phone: (312) 698-2200.

November

Diet and Exercise: Synergism in Health Maintenance, Walt Disney World Complex, Lake Buena Vista, FL, Nov. 3-4. AAFP and AMA Category I credits. Fee: \$60. Contact: Dept. of Foods & Nutrition, AMA, 535 N. Dearborn St., Chicago, IL 60610. Phone: (312) 751-6524.

Computer Tomography Scanning of the Brain, Masur Aud., NIH Clinical Ctr., Bldg. 10, Bethesda, MD, Nov. 4-6. Contact: Dr. Michael D. Walker, Dir., Stroke & Trauma Prog., Nat'l Instit. of Neuro. & Comm. Disorders & Stroke, Fed. Bldg., Rm. 8A08, 7550 Wisconsin Ave. Bethesda, MD 20205. Phone: (301) 496-2581.

AAMI 1981 Regional Meeting, Loews Anatole, Dallas, TX, Nov. 19-20. Fee: \$90 for full day, \$45 for evening & half day. Contact: Renee Pietranglo, AAMI, Suite 602, 1901 N. Ft. Myer Dr., Arlington, VA 22209. Phone: (703) 525-4890.

December

Behavioral Medicine and Primary Care in the '80's, Ilikai Hotel, Honolulu, HI, Dec. 4-11. 16 hrs. AAFP and AMA Category I credits. Fee: \$300. Contact: Jeri McClain, Adm. Assist., USC School of Med., Office of Academic Affairs, Columbia, SC 29208. Phone: (803) 777-7470.

February

Rheumatology Seminar IV, South Seas Plantation Resort, Captiva Island, FL, Feb. 27—Mar. 6. 20 hrs. Category I credits. Fee: \$250. Contact Dept. of CME, MMA, Suite 400, Hlth. Assoc. Center, 2221 University Ave., SE, Minneapolis, MN 55414. Phone: (612) 378-1875.

SOUTH DAKOTA JOURNAL OF MEDICINE

Published Monthly by the S.D. State Medical Assn.

Volume XXXIV September 1981 Number 9



**The Comprehensive Diagnosis Of
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**Clinicopathological Conference
Fifty-Four Year Old Caucasian Male With
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X-Ray Case Of The Month

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Feelings vs.

Some people feel that I am misused and overused and that I'm prescribed too often and for too many kinds of problems.

The FACT is that approximately eight million people, or about 5 percent of the U.S. adult population, will use me during the current year. By contrast, the national health examination survey (1971-1975) found that 25 percent of the U.S. adult population experiences moderate to severe psychological distress. Additionally, studies of patient attitudes revealed that most patients have realistic views regarding the limitations of tranquilizers and a strong conservatism about their use, as evidenced by a general tendency to decrease intake over time. Finally, a six-year, large-scale, carefully conducted national survey showed that the great majority of physicians appropriately prescribe tranquilizers.

Some people feel that patients being treated with anxiolytic drugs are "weak," can't tolerate the anxieties of normal daily living, and should be able to resolve their problems on their own without the help of medication.

The FACT is that while most people can withstand normal, everyday anxieties, some people experience excessive and persistent levels of anxiety due to personal or clinical problems. An extensive national survey concluded that Americans who do use tranquilizers have substantial

Facts

justification as evidenced by their high levels of anxiety. It was further noted that antianxiety drugs are not usually prescribed for trivial, transient emotional problems.

Some people feel afraid of me because of the stories they've heard about my being harmful and having the potential to produce physical dependence.

The FACT is that there are thousands of references in the medical literature documenting my efficacy and safety. Extensive and painstakingly thorough studies of toxicological data conclude that I am one of the safest types of psychotropic drugs available. Moreover, I do not cause physical dependence if the recommended dosage and therapeutic regimen are followed under careful physician supervision. However, I can produce dependence if patients do not follow their physicians' directions and take me for prolonged periods, at dosages that exceed the therapeutic range. Patients for whom I have been prescribed should be cautious about their use of alcohol because an additive effect may result.

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Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal, adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis; stiff-man syndrome, convulsive disorders (not for sole therapy). The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma, may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed, drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other anti-depressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported, should these occur, discontinue drug. Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. **Adults:** Anxiety disorders, symptoms of anxiety, 2 to 10 mg b.i.d. to q.i.d., alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed, adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d., adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. **Geriatric or debilitated patients:** 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) **Children:** 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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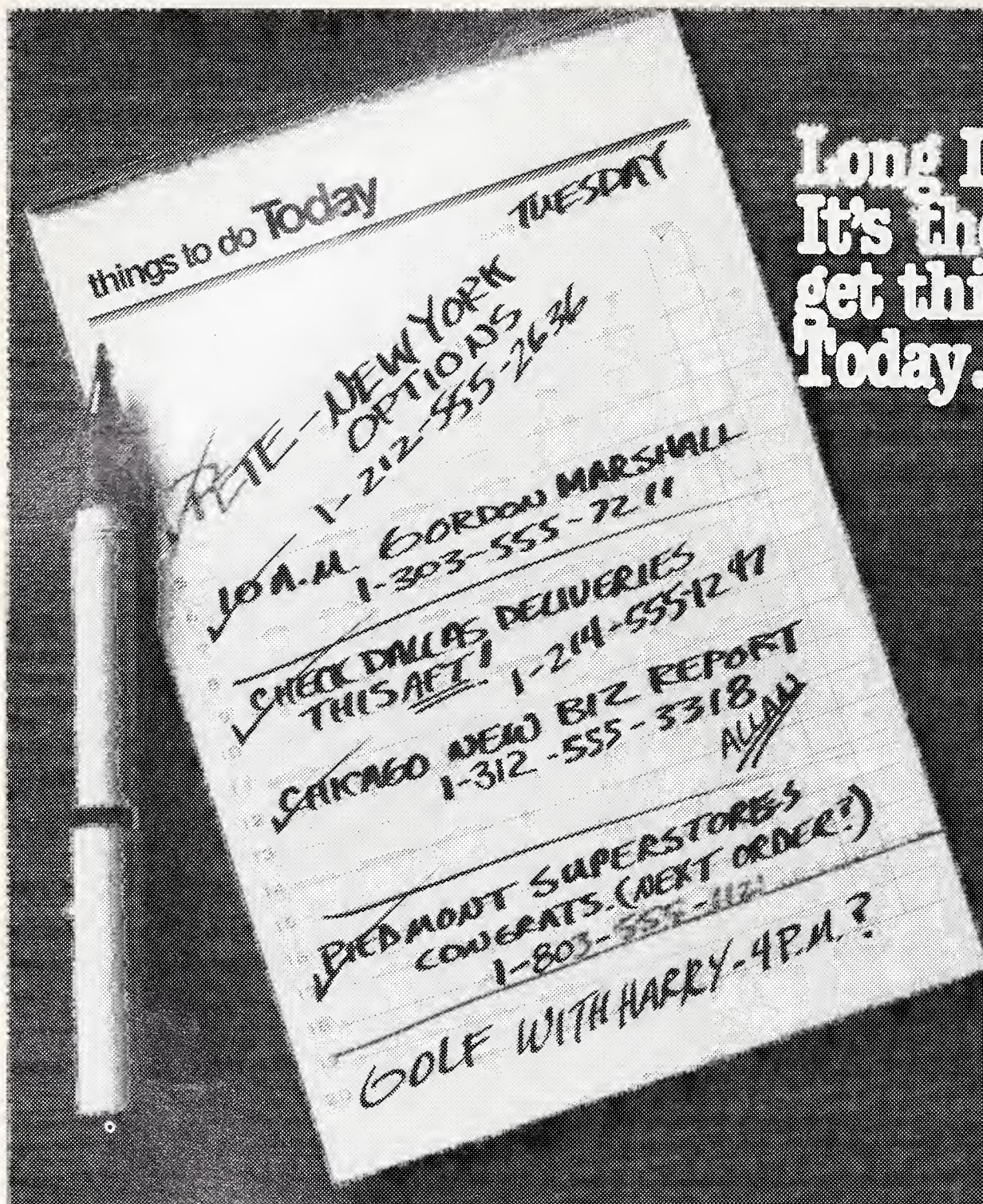
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EQUAGESIC—Abbreviated Summary

INDICATIONS: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

“Possibly” effective for the treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease or tension headache.

Final classification of the less-than-effective indications requires further investigation.

The effectiveness of Equagesic in long-term use, i.e. more than four months, has not been assessed by systematic clinical studies. The physician should periodically reassess usefulness of the drug for the individual patient.

CONTRAINDICATIONS: Equagesic should not be given to individuals with a history of sensitivity or severe intolerance to aspirin, meprobamate, or ethoheptazine citrate.

WARNINGS: Careful supervision of dose and amounts prescribed for patients is advised, especially with those patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g., alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on or habituation to the drug. Where excessive dosage has continued for weeks or months, dosage should be reduced gradually rather than abruptly stopped, since withdrawal of a “crutch” may precipitate withdrawal reaction of greater proportions than that for which the drug was originally prescribed. Abrupt discontinuance of doses in excess of the recommended dose has resulted in some cases in the occurrence of epileptiform seizures.

Special care should be taken to warn patients taking meprobamate that tolerance to alcohol may be lowered with resultant slowing of reaction time and impairment of judgment and coordination.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with the use

of minor tranquilizers (meprobamate, chlordiazepoxide, and diazepam) during the first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physicians about the desirability of discontinuing the drug.

Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breast-feeding patients, the drug's higher concentration in breast milk as compared to maternal plasma levels should be considered.

Preparations containing aspirin should be kept out of the reach of children. Equagesic is not recommended for patients 12 years of age and under.

PRECAUTIONS: Should drowsiness, ataxia, or visual disturbance occur, the dose should be reduced. If symptoms continue, patients should not operate a motor vehicle or any dangerous machinery.

Suicidal attempts with meprobamate have resulted in coma, shock, vasomotor and respiratory collapse, and anuria. Very few suicidal attempts were fatal although some patients ingested very large amounts of the drug (20 to 40 gm). These doses are much greater than recommended. The drug should be given cautiously, and in small amounts, to patients who have suicidal tendencies. In cases where excessive doses have been taken, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Hyperventilation has been reported occasionally. Any drug remaining in the stomach should be removed and symptomatic treatment given. Should respiration become very shallow and slow, CNS stimulants, e.g., caffeine, Metrazol, or amphet-

mine, may be cautiously administered. If severe hypotension develops, pressor amines should be used parenterally to restore blood pressure to normal levels.

ADVERSE REACTIONS: A small percentage of patients may experience nausea with or without vomiting and epigastric distress. Dizziness occurs rarely with meprobamate and ethoheptazine citrate with aspirin administered in recommended dosage. The meprobamate may cause drowsiness but, as a rule, this disappears as therapy is continued. Should drowsiness persist and be associated with ataxia, this symptom can usually be controlled by decreasing the dose, but occasionally it may be desirable to administer central stimulants such as amphetamine or mephentermine sulfate concomitantly to control drowsiness.

A clearly related side effect to the administration of meprobamate is the rare occurrence of allergic or idiosyncratic reactions. This response develops, as a rule, in patients who have had only 1-4 doses of meprobamate and have not had a previous contact with the drug. Previous history of allergy may or may not be related to the incidence of reactions.

Mild reactions are characterized by an itchy urticarial or erythematous, maculopapular rash which may be generalized or confined to the groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema, and fever have also been reported.

More severe cases, observed only very rarely, may also have other allergic responses, including fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case), and hyperthermia. Treatment should be symptomatic such as administration of epinephrine, antihistamine, and possibly hydrocortisone. Meprobamate should be stopped, and re-institution of therapy should not be attempted.

Rare cases have been reported where patients receiving meprobamate suffered from aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia. In nearly every instance reported, other toxic agents known to have caused these conditions have been associated with meprobamate. A few cases of leukopenia during

continuous administration of meprobamate are reported, most of these returned to normal without discontinuation of the drug.

Impairment of accommodation and visual acuity has been reported rarely.

OVERDOSE: Two instances of accidental or intentional significant overdosage with ethoheptazine citrate combined with aspirin have been reported. These were accompanied by symptoms of CNS depression, including drowsiness and light-headedness, with uneventful recovery. However, on the basis of pharmacological data, it may be anticipated that CNS stimulation could occur. Other anticipated symptoms would include nausea and vomiting. Appropriate therapy of signs and symptoms as they appear is the only recommendation possible at this time. Overdosage with ethoheptazine combined with aspirin would probably produce the usual symptoms and signs of salicylate intoxication. Observation and treatment should include induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions.

DESCRIPTION: Each Equagesic tablet contains 150 mg meprobamate, 75 mg ethoheptazine citrate and 250 mg aspirin.

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*This drug has been evaluated as possibly effective for this indication.

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WYGESIC—Abbreviated Summary

INDICATION: For the relief of mild-to-moderate pain.

CONTRAINDICATION: Hypersensitivity to propoxyphene or to acetaminophen.

WARNINGS: CNS ADDITIVE EFFECTS AND OVERDOSAGE: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, or other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. Toxic effects and fatalities have occurred following overdoses of propoxyphene alone or in combination with other CNS depressants. Most of these patients had histories of emotional disturbances or suicidal ideation or attempts, as well as misuse of tranquilizers, alcohol, or other CNS-active drugs. Caution should be exercised in prescribing large amounts of propoxyphene for such patients (see Management of Overdosage).

DRUG DEPENDENCE: Propoxyphene can produce drug dependence characterized by psychic dependence and less frequently physical dependence and tolerance. It will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics. The abuse liability of propoxyphene is qualitatively similar to codeine's although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine.

USAGE IN AMBULATORY PATIENTS: Propoxyphene may impair the mental and/or physical abilities required for potentially hazardous tasks, e.g. driving a car or operating machinery. Patients should be cautioned accordingly.

USAGE IN PREGNANCY: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. **INSTANCES OF WITHDRAWAL SYMPTOMS IN THE NEONATE HAVE BEEN REPORTED FOLLOWING USAGE DURING PREGNANCY.** Therefore, propoxyphene should not be used in pregnant women unless, in the

judgement of the physician, the potential benefits outweigh the possible hazards.

USAGE IN CHILDREN: Propoxyphene is not recommended for children because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric group.

PRECAUTIONS: Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine. The CNS depressant effect of propoxyphene may be additive with other CNS depressants, including alcohol.

ADVERSE REACTIONS: The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting. These seem more prominent in ambulatory than in nonambulatory patients; some of these reactions may be alleviated if the patient lies down. Other adverse reactions include constipation, abdominal pain, skin rashes, light-headedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances. The chronic ingestion of propoxyphene in doses over 800 mg per day has caused toxic psychoses and convulsions. Cases of liver dysfunction have been reported.

DRUG INTERACTIONS: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, and other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended (see Warnings). Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine.

MANAGEMENT OF OVERDOSAGE: SYMPTOMS: The manifestations of serious overdosage with propoxyphene are similar to those of narcotic overdosage and include respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, cyanosis), extreme somnolence progressing to stupor or coma, pupillary constriction, and circulatory collapse. In addition to these characteristics, which are reversed by narcotic antago-

nists such as naloxone, there may be other effects. Overdoses of propoxyphene can cause delay of cardiac conduction as well as focal or generalized convulsions, a prominent feature in most cases of severe poisoning. Cardiac arrhythmias and pulmonary edema have occasionally been reported, and apnea, cardiac arrest, and death have occurred.

Symptoms of massive overdosage with acetaminophen may include nausea, vomiting, anorexia, and abdominal pain, beginning shortly after ingestion and lasting for 12 to 24 hours. However, early recognition may be difficult since early symptoms may be mild and nonspecific. Evidence of liver damage is usually delayed. After the initial symptoms, the patient may feel less ill; however, laboratory determinations are likely to show a rapid rise in liver enzymes and bilirubin. In case of serious hepatotoxicity (jaundice, coagulation defects, hypoglycemia, encephalopathy, coma, and death may follow. Renal failure due to tubular necrosis, and myocardiopathy, have also been reported.

Ingestion of 10 grams or more of acetaminophen may produce hepatotoxicity. A 13-gram dose has reportedly been fatal.

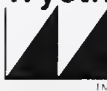
TREATMENT: Primary attention should be given to the reestablishment of adequate respiratory exchange through provision of a patent airway and institution of assisted or controlled ventilation. The narcotic antagonists, naloxone, nalorphine, and levallorphan, are specific antidotes against the respiratory depression produced by propoxyphene. An appropriate dose of one of these antagonists should be administered preferably I.V., simultaneously with efforts at respiratory resuscitation and the antagonist should be repeated as necessary until the patient's condition remains satisfactory. In addition to a narcotic antagonist, the patient may require careful titration with an anticonvulsant to control seizures. Analeptic drugs (e.g. caffeine or amphetamine) should not be used because of their tendency to precipitate convulsions.

Oxygen, IV fluids, vasopressors and other supportive measures should be used as indicated. Gastric lavage may be helpful. Activated charcoal can absorb a significant amount of ingested propoxyphene. Dialysis is of little value in poisoning by propoxyphene alone. Acetaminophen is rapidly absorbed and efforts to remove the drug from the body should not be delayed. Copious gastric lavage and/or induction of emesis may be indicated. Activated charcoal is probably ineffective unless administered almost immediately after acetaminophen ingestion. Neither forced diuresis nor hemodialysis appears to be effective in removing acetaminophen. Since acetaminophen in overdose may have an antidiuretic effect and may produce renal damage, administration of fluids should be carefully monitored to avoid overload. It has been reported that mercaptamine (cysteine) or other thiol compounds may protect against liver damage if given soon after overdosage (8-10 hours). N-acetylcysteine is under investigation as a less toxic alternative to mercaptamine, which may cause anorexia, nausea, vomiting, and drowsiness. Appropriate literature should be consulted for further information (JAMA 237 2406-2407, 1977).

Clinical and laboratory evidence of hepatotoxicity may be delayed up to one week. Acetaminophen plasma levels and half-life may be useful in assessing the likelihood of hepatotoxicity. Serial hepatic enzyme determinations are also recommended.

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The Comprehensive Diagnosis Of Developmental Behavioral Disorders In Primary Care: An Integrated Approach

Vinod Bhatara, M.D., M.S., F.R.C.P.(C)*

ABSTRACT

Coexistence and overlap of multiple dimensions of dysfunctions (developmental, behavioral and physical) in childhood requires a multidimensional assessment of cases for ideal intervention; i.e., multimodality treatment.

The primary care physician can initiate comprehensive assessment of childhood behavioral-developmental dysfunction by a thorough history and physical examination. By pinpointing problems in multiple dimensions (physical, developmental, and behavioral), the physician may facilitate further multi-faceted evaluation. He or she may choose to

Primary care physicians are often the first ones to see the highly prevalent disorders of childhood: developmental and/or behavioral problems. Physician training needs in such areas as learning disorders are being increasingly recognized. Following is the consensus reached at a recent medical symposium on the need for interdisciplinary treatment of learning problems.¹ "Primary care physicians can be helpful to these children but need considerably more information in such problems." According to a survey reported at the above symposium, 73 percent of pediatricians in the sample felt the "need for more training in diagnosis and treatment of learning disabilities." Adding to this problem of inadequate training are several other sources of di-

make appropriate referrals (e.g. psychological testing, pediatric neurology) and/or collect further data (e.g. developmental screening tests, playroom observations) for final and definitive diagnoses. An approach to multidimensional (multiaxial) diagnosis is outlined.

This article also highlights two sources of diagnostic confusion in diagnosing behavioral-developmental dysfunction: overlapping physical, behavioral, and developmental diagnoses and semantics. Diagnostic refinements contained in the latest revision of the Diagnostic and Statistical Manual (DSM-III) of Mental Disorders are discussed to reduce confusion from these two sources.

agnostic and conceptual confusion: developmental variance; semantics; and overlap of behavioral, developmental and neurological problems. Given the wide range of variance in the rate and sequence of development (especially in the early childhood), firm statements about diagnosis, prognosis and treatment are difficult to make. Yet early detection is important for specific remedial treatment and for prevention of such psychosocial sequelae of childhood disorders as poor peer relationship, withdrawal, low self-esteem, school failure, and delinquency. Using the common childhood problems of hyperactivity and learning disorders as examples, the other two areas of conceptual confusion (semantics and problem of overlapping diagnoses) are discussed below.

Semantics

Clinicians from different professions (varying with the purpose of their assessment) appear to evaluate

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patients from different² conceptual dimensions. Thus, one clinician may only focus on a child's hyperactivity (behavioral axis), another may only be interested in minimal brain dysfunction³—MBD (neurological axis). Yet another clinician may only be aware of the learning disability (cognitive or developmental axis). Not only has this tendency resulted in the semantic confusion, but also in diagnostic confusion. As a way out of this conceptual maze, this article uses the refinements and terminology adopted by the latest edition of the **Diagnostic and Statistical Manual of Mental Disorders (DSM-III)**. The authors of DSM-III took the stance of adopting non-etiological, descriptive terms (because etiology is not known at present) which emphasized the "core behavioral symptom:" Attention Deficit Disorder (ADD). Hyperactivity or hyperkinesis was dropped as a main term. This is because there are several patients (particularly the adolescents) who have problems at attention but have no activity problems. Problems of learning are now diagnosable separately from developmental and relevant physical problems. Popular diagnostic terms in the behavioral, developmental, and neurologic dimension and their DSM-III equivalents are shown in Table I. (In-depth discussion of the multi-axial diagnostic system can be found in DSM-III⁴.)

Overlapping Behavioral, Developmental and Neurological Diagnoses

As Figure 1 represents, neurological conditions (MBD and other central nervous dysfunctions) often overlap with the behavioral concepts (ADD) and developmental-cognitive concepts of learning disability. To emphasize this overlap of behavior and development, the use of the term behavioral-developmental disorders is used in this article. The extent of this problem of overlapping behavioral

and developmental dysfunction in private practice may be judged from a study by Oberklaid and Associates⁵ involving 79 children. Of the 37 children referred for behavioral concerns, at least 13 (more than a third of those with behavioral problems) had undetected developmental problems requiring specific interventions. Thus, the area of predominant disturbance (behavior, CNS or development) is often difficult to determine. This is because behavioral and developmental dysfunctions (e.g. hyperactivity and learning disability) are not mutually exclusive dysfunctions. As Figure 1 represents, usually the overlap exists horizontally. Thus, most frequent association is between the concepts of attention-deficit disorder (ADD), MBD, and learning disability (LD). While most children with ADD are also learning disabled,⁶ LD may affect a larger number of children (perhaps 10 to 20 percent in contrast to the prevalence of ADD, which may be about 3 to 5 percent). Similarly, most autistic children are also mentally² retarded. Although Figure 1 emphasizes overlap in the horizontal direction (perhaps the most frequent overlap), the overlapping diagnostic problems exist vertically (e.g. between MBD and cerebral palsy)⁷ and even diagonally (some autistics may have learning disabilities). Further, each of the conditions may exist independently of the overlapping condition. Thus, LD may exist without MBD or ADD. Just like CNS dysfunction associated with autism, MBD may not be a homogenous condition. This problem of overlapping diagnoses and semantic confusion has been further illustrated by Table I.

This article suggests that frequently coexistence and overlap of multiple dimensions of dysfunctions (physical, behavioral and developmental) in childhood requires a multi-dimensional assessment of cases for the ideal intervention, i.e. multimodality treatment. Using the very common childhood prob-

Table I
Problem of Overlapping Diagnoses, and the Need for
Multiaxial (multidimensional) Evaluation of Hyperactivity and/or Learning Disability

Table I Problem of Overlapping Diagnoses, and the Need for Multiaxial (multidimensional) Evaluation of Hyperactivity and/or Learning Disability						
DSM-III Axis	I		II		III	
Diagnostic Dimension	Behavioral		Developmental		Neurologic	
Source of Diagnostic Terms	Popular Terms	Equivalent DSM-III Terms	Popular Terms	Equivalent DSM-III Terms	Popular Terms	DSM-III Recommended Terms
Overlapping Diagnostic Terms (concepts)	Hyperactivity, Hyperkinesis, Hyperkinetic reaction	Attention deficit disorder, with hyperactivity (ADD) (Also recognized is the existence of attention deficit disorder, without hyperactivity and attention deficit disorder, residual type)	Learning Disability (LD)	Specific developmental disorders —developmental reading disorder —developmental arithmetic disorder —developmental language disorder —developmental articulation disorder —mixed specific developmental disorder	Minimal brain dysfunction (MBD) —with identifiable and etiologically relevant cause (lead ingestion, infection, etc.) —of unknown origin	Diagnostic terms from ICD-9-CM** (but outside the mental disorders section) Appropriate physical findings (for example soft neurological signs) may be recorded
Usually the overlap occurs among the conditions as grouped (i.e., in the horizontal direction). But, rarely other overlaps may occur e.g., an autistic may have a Learning Disability. Thus, the possible presentations include: ADD without LD or MBD, ADD+LD+MBD, ADD+LD, ADD+MBD, MBD+LD without ADD, MBD without LD or ADD, and LD without MBD or ADD. International Classification of Diseases, 9th Edition, Clinical Modification.						

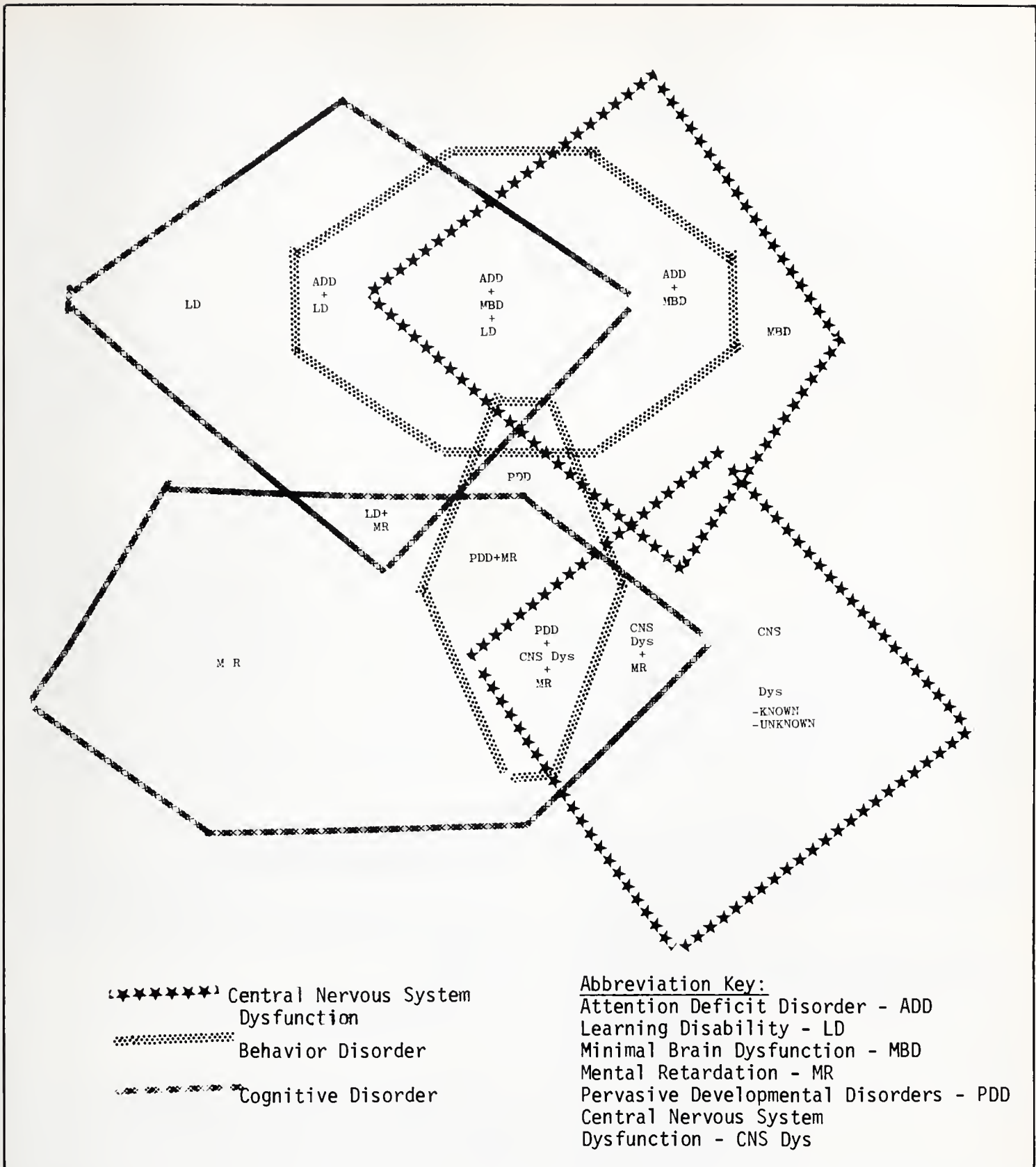


Figure 1

Overlapping physical, behavioral, and cognitive diagnostic concepts in behavioral-developmental disorders of childhood.

blems of learning and hyperactivity as examples, an outline for such a multidimensional (multiaxial) assessment is provided.

Assessment of Child with Problems of Learning and/or Behavior: An Integrated Approach

There is a need to develop a systematic method of integrating multiple dimensions in management

of health problems.⁸ Utilizing the concepts presented above, it is possible to derive the following rational assessment procedure.

A. A good history and physical examination remain the best tools for initiating the diagnostic process.

B. Physician should look for conditions which frequently coexist or overlap with the given problem

e.g. in case of a child referred for a learning problem, overlapping diagnostic conditions can be systematically ruled in or out one-by-one in the following order:

1. Such CNS dysfunctions as MBD and other focal or nonfocal neurological deficits
2. Mental retardation
3. Because of the frequent association between LD and ADD, physicians should look for ADD in all cases of LD and vice-versa. According to one estimate⁶, as much as 80% of the children referred for learning problems may have ADD.
4. Specific developmental disorders are delays in any one or more of these areas: reading, arithmetic, language (receptive or expressive) or articulation.
5. Conduct disorders are also commonly associated with LD.
6. Any other problems e.g. such emotional difficulties secondary to the learning problems as anxiety and low self esteem.

C. Multiaxial Assessment

By pinpointing problems in multiple dimensions (physical, developmental, and behavioral) the physician can facilitate further multifaceted evaluation. He or she may choose to make appropriate referrals (e.g. psychological testing, pediatric neurology, child psychiatry) and/or collect further data (e.g. devel-

opmental screening tests, playroom observations) for final and definitive diagnoses. An approach to DSM-III multiaxial assessment (in case of a hyperactive and learning disabled child) is outlined. With the full appreciation that both the behavioral and developmental conditions may be the products of brain pathology, from practical considerations it may be useful to consider assessments in different areas separately. Thus, Table II illustrates physical evaluation (DSM-III, axis III) evaluation and Tables III and IV behavioral (DSM-III, axis I) and specific developmental (DSM-III, axis II) assessments respectively. In addition to a thorough history and physical examination, the approach outlined in Table II suggests an expanded data base (including questionnaires) to identify various tip-offs for an early recognition of a developmental-behavioral disorder such as hyper-activity (attention deficit disorder).

Although a firm diagnosis is not recommended before the age of five years, early recognition is important. Visual defects, hearing loss, interpersonal problems, and other difficulties may be discernable by one year of age^{9,10}. Table II lists several identifiable medical-neurological "flags" of at-risk status. A large number of these features is very likely to correlate with a CNS dysfunction (or associated ADD).

The physician should clarify the neurologic status of the child. The physician should assess the history

Table II
**Medical-Neurologic (AXIS-III) Evaluation of a Child With Behavioral-
Developmental Dysfunctions: Pointers Towards at Risk Status**

SOURCES OF DATA BASE:	Parent filled medical-developmental questionnaire, historical information obtained from the parents, repeated Denver developmental Screening Test, Physical Examination (including height, weight and head circumference, Vision and hearing (including audiometric screening), and Developmental Neurological Examination, Preschool Teacher Questionnaire.
<u>TIME PERIOD OF CLINICAL DATA—SOME TYPICAL FEATURES</u>	
Pregnancy (fetus)	First trimester bleeding, chemical (including alcohol and tobacco use), rubella, poor nutrition, or any other possible source of stress on the fetal CNS.
Birth	Prolonged labor, placenta previa, prematurity; small for term weight (less than 5½ lb.); or any other cause of CNS hypoxia.
Infancy	1) Such severe CNS illness as meningitis, lead encephalopathy; 2) history of head trauma (crib fall); 3) persistent colic; 4) hyperactivity (high pitched cry, difficulties in sucking, sleeping, swallowing and feeding, difficult temperament, overreactive to various stimuli); 5) hyporeactivity (hypotonia, lethargy, low activity or a combination of 6) hypo- and hyper-reactivity; 7) delayed development; 8) visual deficit; 9) hearing deficit; 10) labile responses especially in the mother-child interaction; 11) lack of responsiveness to others, e.g. poor cuddliness.
Toddler	As above, especially persistent hyperreactivity, childhood illnesses, and sleep problems. May show "paradoxical" irritant effect of barbiturates. Motor activity, especially exploratory activity, may be exaggerated and excessive. History of "mile stones" may show lagging development.
Preschool	As above. In addition, data may be available from teachers. Behaviorally look for inattention, impulsivity and hyperactivity.
Other data	Family history may be relevant in disorders with heredo-familial tendency (e.g. some types of mental retardation, hyperactivity). Specific developmental disorders (Axis II) in language, speech, may occur. Sensitivity to chocolates, additive & some foodstuffs (allergic tension fatigue syndrome) and other treatable causes of the disorder (e.g. lead, toxins, etc.) should be considered. EEG is obtained, if a seizure disorder is suspected.

Table III (DSM-III—Axis I Evaluation)
DSM-III DIAGNOSTIC CRITERIA FOR ATTENTION DEFICIT DISORDER WITH HYPERACTIVITY

Typical Signs	
Developmentally Inappropriate Signs	Operational criteria are given below for a child older than eight (8) years. Number of criteria required for diagnosis is greater than the number specified below in a younger child.
Inattention	At least three (3) of the following: (1) often fails to finish things he/she starts (2) often doesn't seem to listen (3) easily distracted (4) has difficulty concentrating on schoolwork or other tasks requiring sustained attention (5) has difficulty sticking to a play activity
Impulsivity	At least three (3) of the following: (1) often acts before thinking (2) shifts excessively from one activity to another (3) has difficulty organizing work (exclude difficulties due to cognitive impairment) (4) needs a lot of supervision (5) frequently calls out in class (6) has difficulty awaiting turn in games or group situations
Hyperactivity	At least two (2) of the following: (1) runs about or climbs on things excessively (2) has difficulty sitting still or fidgets excessively (3) has difficulty staying seated (4) moves about excessively during sleep (5) is always "on the go" or acts as if "driven by a motor"
Other features (onset, duration, and exclusion criteria)	Onset before the age of seven (7) years Duration of at least six months Not due to schizophrenia, affective disorder, or severe or profound mental retardation

from pregnancy on for the evidence of an event causing CNS dysfunction (eg. encephalitis). Diagnosable neurological dysfunction often relieves parents' guilt related to the child's difficulties. The physician should rule out such chronic CNS dysfunctions as seizure disorders and cerebral palsy (associated with ADD). Thus, EEG should be evaluated primarily for the evidence of a seizure disorder. If a seizure disorder is found, barbiturates and related drugs should be avoided because these drugs may worsen the clinical condition. Cerebral palsy may be identifiable by such problems as spastic diplegia and spastic hemiplegia. If cerebral palsy is recognizable, it establishes the presence of a previous CNS dysfunction. It implies attention to the child's motor and possible language difficulties.

Of course, it may not be possible to provide services for all these children at the primary care level. Referral (in case of doubt) may be made to specialists in such fields as pediatric neurology, child psychiatry, or educational psychology.

Table III lists the refined diagnostic criteria for attention deficit disorders with hyperactivity from DSM-III. A major advance contained in these DSM-III criteria is that these criteria are operational.

It is important to note that behavioral signs of attention deficit disorder (ADD) may not be observable in a physicians office. Thus, the following sources of data base in the process of diagnosis is used. The sources of a data base may be many: behavioral questionnaires and rating scales com-

pleted by the parents and teacher, psychoeducational testing from preschool if available, family and child interviews, and direct behavior observation if necessary. Teacher ratings of behavior are more objective than the parental rating scales. Connors' Abbreviated Rating Scale¹¹ is the scale most commonly used. Normal range of score is available for this scale in school-age children. Another feature of this scale is that the symptom clusters in the scale are drug sensitive (the ratings indicate lower post-drug than pre-drug score in a stimulant responder). However, if doubt about the diagnosis exists, the physician may (1) refer for further evaluation, (2) be involved in direct behavioral observation because the symptoms are typically variable. Observation in situations which are task-oriented (e.g. group situations, tasks requiring sustained attention or pre-school classroom) may be necessary. (3) Further, a child may suffer from ADD without hyperactivity (as DSM-III recognizes).

Table IV summarizes one approach¹² to the developmental assessment of a child presenting with learning problems.

D. If physician suspects family or environmental problems appropriate referral should be made. A discussion about the assessment of family is beyond the scope of this paper but is of paramount importance.

Finally, it is important to point out that children's serious learning and/or behavior problems are best treated and evaluated by an interdisciplinary team acting in concert. The approach outlined in this

<div>Table IV</div> <div>*SPECIFIC DEVELOPMENTAL (DSM III) EVALUATION OF BEHAVIORAL-DEVELOPMENTAL DYSFUNCTION</div>		
Data Base	Data Sought	Assessment from the Data
<ul style="list-style-type: none"> —History —Parent filled developmental questionnaires —Teacher filled questionnaires 	Specific developmental delays	History of gross and specific delays in such areas as reading, writing, language, articulation or motor function. Problems may be present in more than one of the areas mentioned above (mixed type of specific developmental disorder).
<ul style="list-style-type: none"> —Repeated Denver developmental screening tests 	Quick screening for developmental delays	Because "the brevity of instrument bears a direct relationship to its unreliability", DDST should be considered only a screening instrument for population at risk. This test will not define the problem of a specific child in sufficient depth to be therapeutically relevant.
<ul style="list-style-type: none"> —Playroom observation 	Problems in processing information in the following areas: <ul style="list-style-type: none"> —Input —Integration —Memory —Output 	<ul style="list-style-type: none"> —Problems in sensory input (especially auditory or visual perception) may be deduced. For example, during the play session a child overuses one sensory modality. He may have difficulty in organizing input as shown by bumping into things. Visual-motor dysfunction may be reflected by more than normal errors in copying geometric shapes (or by a formal Bender-Gestalt test). Auditory perceptual problems may be manifested by apparent difficulty in listening or understanding. There may also be intersensory problems. —Problems in integrating information may be shown by difficulties in sequencing (e.g. child may start a story in the middle), or age-appropriate abstraction (e.g. in generalizing from a story). —Problems of short term memory (assessed by requiring the child to remember unrelated words after 5 minutes) or long term memory (ability to remember events of relatively remote past). —Problems of output (as shown by language output or motor output) e.g. inconsistencies in language, clumsiness in walking (gross motor disability), eye-hand coordination (playing basketball), or poor penmanship (fine motor disability).
<ul style="list-style-type: none"> —Neurological examination 	<ul style="list-style-type: none"> —Focal signs —Soft signs 	Frequently, neurological evaluations are useful in finalizing diagnosis. Assessment from soft signs may be of questionable value (there is no localizing value).
<ul style="list-style-type: none"> —Specific psychological and educational testing 	One or more areas of learning disorder	Necessary for final and definitive diagnosis. It is preferable to make referral to an educational psychologist with a specific question (rather than recommendation of a specific test). An intellectual assessment (e.g. Wechsler Intelligence Scale for Children) can not only rule out mental retardation, but also provides useful information in the areas of poor score. Verbal tests may correlate well with the function of dominant cortical hemisphere and the performance with non-dominant hemisphere.
*The approach outlined is rather simplified and is by no means exhaustive, e.g. It does not include learning problems caused by selective attention or impulsivity.		

paper can only facilitate interdisciplinary approach and is not meant to replace assessments by multiple disciplines. However, only a physician is in a special position to realize that a stimulant may affect behavior, development, and learning differentially. He or she may thus be able to cut across disciplines with the overall purpose of tailoring an individualized multimodal treatment plan to the needs of the star of the therapeutic team—the child.

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INDICATIONS AND USAGE

For the prevention and treatment of nocturnal recumbency leg muscle cramps.

CONTRAINDICATIONS

Quinamm may cause fetal harm when administered to a pregnant woman. Congenital malformations in the human have been reported with the use of quinine, primarily with large doses (up to 30 g) for attempted abortion. In about half of these reports the malformation was deafness related to auditory nerve hypoplasia. Among the other abnormalities reported were limb anomalies, visceral defects, and visual changes. In animal tests, teratogenic effects were found in rabbits and guinea pigs and were absent in mice, rats, dogs, and monkeys. Quinamm is contraindicated in women who are or may become pregnant. If this drug is used during pregnancy, or if the patient becomes pregnant while taking this drug, the patient should be apprised of the potential hazard to the fetus. Because of the quinine content, Quinamm is contraindicated in patients with known quinine hypersensitivity and in patients with glucose-6-phosphate dehydrogenase (G-6-PD) deficiency.

Since thrombocytopenic purpura may follow the administration of quinine in highly sensitive patients, a history of this occurrence associated with previous quinine ingestion contraindicates its further use. Recovery usually occurs following withdrawal of the medication and appropriate therapy.

This drug should not be used in patients with tinnitus or optic neuritis or in patients with a history of blackwater fever.

WARNINGS

Repeated doses or overdosage of quinine in some individuals may precipitate a cluster of symptoms referred to as cinchonism. Such symptoms, in the mildest form, include ringing in the ears, headache, nausea, and slightly disturbed vision, however, when medication is continued or after large single doses, symptoms also involve the gastrointestinal tract, the nervous and cardiovascular systems, and the skin.

Hemolysis (with the potential for hemolytic anemia) has been associated with a G-6-PD deficiency in patients taking quinine. Quinamm should be stopped immediately if evidence of hemolysis appears.

If symptoms occur, drug should be discontinued and supportive measures instituted. In case of overdosage, see OVERDOSAGE section of prescribing information.

PRECAUTIONS

General
Quinamm should be discontinued if there is any evidence of hypersensitivity (See CONTRAINDICATIONS). Cutaneous flushing, pruritus, skin rashes, fever, gastric distress, dyspnea, ringing in the ears, and visual impairment are the usual expressions of hypersensitivity, particularly if only small doses of quinine

have been taken. Extreme flushing of the skin accompanied by intense, generalized pruritus is the most common form. Hemoglobinuria and asthma from quinine are rare types of idiosyncrasy.

In patients with atrial fibrillation, the administration of quinine requires the same precautions as those for quinidine. (See Drug Interactions.)

Drug Interactions

Increased plasma levels of digoxin and digitoxin have been demonstrated in individuals after concomitant quinidine administration. Because of possible similar effects from use of quinine, it is recommended that plasma levels for digoxin and digitoxin be determined for those individuals taking these drugs and Quinamm concomitantly.

Concurrent use of aluminum-containing antacids may delay or decrease absorption of quinine.

Cinchona alkaloids, including quinine, have the potential to depress the hepatic enzyme system that synthesizes the vitamin K dependent factors. The resulting hypoprothrombinemic effect may enhance the action of warfarin and other oral anticoagulants.

The effects of neuromuscular blocking agents (particularly pancuronium, succinylcholine, and tubocurarine) may be potentiated with quinine, and result in respiratory difficulties.

Urinary alkalinizers (such as acetazolamide and sodium bicarbonate) may increase quinine blood levels with potential for toxicity.

Drug Laboratory Interactions

Quinine may produce an elevated value for urinary 17-ketogenic steroids when the Zimmerman method is used.

Carcinogenesis, Mutagenesis, Impairment of Fertility

A study of quinine sulfate administered in drinking water (0.1%) to rats for periods up to 20 months showed no evidence of neoplastic changes.

Mutation studies of quinine (dihydrochloride) in male and female mice gave negative results by the micronucleus test. Intraperitoneal injections (0.5 mM/kg) were given twice, 24 hours apart. Direct *Salmonella typhimurium* tests were negative, when mammalian liver homogenate was added; positive results were found.

No information relating to the effect of quinine upon fertility in animal or in man has been found.

Pregnancy

Category X. See CONTRAINDICATIONS.

Nonteratogenic Effects

Because quinine crosses the placenta in humans, the potential for fetal effects is present. Stillbirths in mothers taking quinine have been reported in which no obvious cause for the fetal deaths was shown. Quinine in toxic amounts has been associated with abortion. Whether this action is always due to direct effect on the uterus is questionable.

Nursing Mothers

Caution should be exercised when Quinamm is given to nursing women because quinine is excreted in breast milk (in small amounts).

ADVERSE REACTIONS

The following adverse reactions have been reported with Quinamm in therapeutic or excessive dosage. (Individual or multiple symptoms may represent cinchonism or hypersensitivity.)

Hematologic: acute hemolysis, thrombocytopenic purpura, agranulocytosis, hypoprothrombinemia.

CNS: visual disturbances, including blurred vision with scotomata, photophobia, diplopia, diminished visual fields, and disturbed color vision, tinnitus, deafness and vertigo, headache, nausea, vomiting, fever, apprehension, restlessness, confusion, and syncope.

Dermatologic: allergic, cutaneous rashes (urticarial, the most frequent type of allergic reaction, papular or scarlatinial), pruritus, flushing of the skin, sweating, occasional edema of the face.

Respiratory: asthmatic symptoms.

Cardiovascular: anginal symptoms.

Gastrointestinal: nausea and vomiting (may be CNS-related), epigastric pain.

DRUG ABUSE AND DEPENDENCE

Tolerance, abuse, or dependence with Quinamm has not been reported.

OVERDOSAGE

See prescribing information for a discussion on symptoms and treatment of overdose.

DOSAGE AND ADMINISTRATION

1 tablet upon retiring. If needed, 2 tablets may be taken nightly—1 following the evening meal and 1 upon retiring.

After several consecutive nights in which recumbency leg cramps do not occur, Quinamm may be discontinued in order to determine whether continued therapy is needed.

Product Information as of October 1980

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WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as a failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of non-susceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section). Complete literature available on request from Professional Services Dept. PML.



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Dr. John Tidd, of Yankton, was honored by the staff of Landmann-Jungman Memorial Hospital for his eleven years of service as the hospital's consulting pathologist.

* * * *

Central Plains Clinic, Sioux Falls, recently announced that **Walter G. Drymalski, M.D.** joined their staff. Dr. Drymalski is a native of Chicago, Ill. He received his degree in medicine from Stritch-Loyola Medical School in Maywood, Ill. and went on to complete a three-year residency in internal medicine at Southwest Michigan Area Health Education Center in Kalamazoo, Mich. Dr. Drymalski is married and has three children.

* * * *

B. T. Brookman, M.D., Wagner, was chosen Citizen of the Year for Wagner. Dr. Brookman came to Wagner in 1952 and has practiced there since that time. He is president of the Wagner Rotary Club, a member of the Chamber of Commerce, Masonic Lodge, S.D. Medical Association and American Medical Association.

* * * *

B. O. Lindbloom, M.D., Pierre, recently spent a week at the University of Wisconsin as the Visiting Fellow in High Risk Obstetrics.

The Madison Clinic, Madison, takes pleasure in announcing the association of **Dr. Richard G. Sample**. Dr. Sample was born in Yankton. He received his B.S. degree from the South Dakota State University in pharmacy in 1972 and spent two years in the army. He then attended the USD School of Medicine receiving his M.D. degree in 1978. He completed a three-year residency program in family practice this year. Dr. Sample and his wife Barbara, a Sioux Falls native, have three children.

(continued)

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Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg; bismuth subgallate, 22.5 mg; bismuth resorcin compound, 17.5 mg; benzyl benzoate, 12.0 mg; Peruvian balsam, 18.0 mg; zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water.

Anusol-HC Suppositories and Anusol-HC Cream help to relieve pain, itching and discomfort arising from irritated anorectal tissues. These preparations have a soothing, lubricant action on mucous membranes, and the antiinflammatory action of hydrocortisone acetate in Anusol-HC helps to reduce hyperemia and swelling.

The hydrocortisone acetate in Anusol-HC is primarily effective because of its antiinflammatory, antipruritic and vasoconstrictive actions.

Indications and Usage: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain, itching and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas, pruritus ani and relief of local pain and discomfort following anorectal surgery.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

Contraindications: Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

Precautions: General: Symptomatic relief should not delay definitive diagnoses or treatment.

Prolonged or excessive use of corticosteroids might produce systemic effects.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Anusol-HC is not for ophthalmic use.

Pregnancy

See "WARNINGS"

Pediatric Use

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

Dosage and Administration: Anusol-HC Suppositories—

Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain comfort with regular Anusol Suppositories.

Anusol-HC Cream—Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

How Supplied: Anusol-HC Suppositories—boxes of 12 (N 0071-1089-07) and boxes of 24 (N 0071-1089-13) in silver foil strips with Anusol-HC printed in black.

Anusol-HC Cream—one-ounce tube (N 0071-3090-13) with plastic applicator.

Store between 59°-86°F (15°-30°C).
1089C010

Dr. Hiroo Kapur, Huron, has earned her board certification from the American Board of Pediatrics. Dr. Kapur received her medical degree at the University of Rajasthan in Jaipur, India.

* * * * *

Southern Hills General Hospital, Hot Springs, has announced the appointment of **Daryl Dickson, M.D.** to its staff. Dr. Dickson is a graduate of USD School of Medicine and has been practicing the last two years as an emergency room physician at Sioux Valley Hospital in Sioux Falls.

* * * * *

Brooks Ranney, M.D., FACOG, of Yankton, a Founding Fellow of the American College of Obstetricians and Gynecologists, was named president-elect of the organization for a one-year term at the group's 29th Annual Clinical Meeting in Las Vegas.

* * * * *

The American College of Physicians announced that **Dr. John Barker,** a gastroenterology specialist and **Dr. Loren Tschetter,** an internal medicine specialist, both of Sioux Falls, have been elected to Fellowship in the national medical specialty society.

* * * * *

Albin Janusz, M.D., Aberdeen, has been elected to a one-year term as president of the South Dakota Chapter of American College of Surgeons.

* * * * *

Wayne R. Shaw, M.D. is the new medical director at the Northeastern Mental Health Center in Aberdeen. Dr. Shaw comes to Aberdeen from Minnesota where he was chief of service at Rochester State Hospital. In addition, he was also a consultant to an alcohol and drug abuse treatment unit in Albert Lea, Minn. along with being in private practice. Dr. Shaw is originally from Tripp, S.D. He received his B.A. degree at the University of South Dakota and his M.D. from the University of Louisville in Kentucky. His psychiatric residency was completed at Austin State Hospital in Austin, Texas where he continued to work for five years at the medical school. He also has a specialty in chemical abuse. He and his wife have two daughters.

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Dennis D. Knutson, M.D., dermatologist, Sioux Falls, has been appointed to serve on the Medical and Scientific Committee for the Dermatology Foundation in 1981. The committee of this national foundation functions to review applications for awards and grants provided by the Dermatology Foundation and determines the recipient of these grants.

Central Plains Clinic, Sioux Falls, announced that Timothy E. Hurley, M.D. will join the Dept. of Internal Medicine. Dr. Hurley was born in Aurora, Ill. He received his pre-med education at the University of North Dakota. He attended medical school at the University of North Dakota and Loyola University. He served an internship and completed his internal medicine residency program at the Mayo Graduate School of Medicine in Rochester, Minn.

Daryl S. Larke, M.D. is a new orthopedic surgeon at Central Plains Clinic, Sioux Falls. Dr. Larke is originally from Battle Creek, Mich. He received his B.A. degree from Kalamazoo College in Michigan and his M.D. degree from Northwestern University School of Medicine. Since then he has been at the Cleveland Clinic Hospital in Cleveland, Ohio, completing a rotating surgical internship and a four year residency in orthopedic surgery.

Bernard King, M.D. has recently moved with his wife and two boys to Salem where he will begin his new practice. Dr. King graduated from Notre Dame University in Indiana in 1976 and then attended the University of South Dakota Medical School for two years and received his M.D. degree from the Washington Medical School in St. Louis, Mo in 1980. He served one year internship at Indiana University Medical School.

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S *President's Page* **D**

Just An Idea!

How many times have you had what you thought was a good idea, only to have it receive less than enthusiastic support when shared over morning coffee with your friends?

Well, hang on, grab a cup of coffee and listen to this idea—

Why not share our Medical School with Wyoming?

Before you choke on your coffee, let me elaborate a bit.

It seems to me that we may have room in our future freshman classes at USD School of Medicine for some Wyoming students and still provide an adequate number of positions for South Dakota in the future. USD School of Medicine admits 65 students to each freshman class and graduates 50 seniors each year. Fifteen sophomores transfer to other schools to complete their MD degree. Doctor Loren Amundson's article in this Journal this past June, concerning Family Practice needs in 1990, indicated a need of an additional 19 Family Physicians per year in South Dakota during the next 10 years to obtain a satisfactory doctor-patient ratio in Family Medicine by 1990. There are no existing studies on total physician needs in South Dakota that I am aware of. Using rough estimations, it would seem that 45 to 50 graduates from our School of Medicine each year would provide the state with adequate future physicians.

This would leave space for 15 to 20 Wyoming students in each entering class. Currently, I understand 30 Wyoming students attend the University of Utah School of Medicine and Creighton School of Medicine on contracts funded largely by the state of Wyoming tax dollars.

By working together with Wyoming, we could develop additional options for all students in the Junior and Senior years as well as possibly providing

funds to enlarge the western campus at Rapid City, etc. There could be advantages to everyone concerned.

With the quality medical school we have in South Dakota, this may be the time to invite Wyoming to send students in the future. South Dakota Medical School officials state that it would save very few tax dollars to reduce enrollment in our own school. So, it makes sense to me to explore this idea for the future—perhaps the near future, remembering that entering freshmen in 1982 would not be completing their residencies until 1989 or 1990.

Well, that's the idea. As you finish your coffee—you can discuss the merits of this idea, if any, or you can spend a few minutes discussing the ridiculous way your president managed to fill this page this month.

Very sincerely yours,



Bruce Lushbough, M.D., President
South Dakota State Medical Association

P.S. I neglected to mention that some exploratory meetings have already been held to explore this concept, so the idea is not totally original. I am sure Dr. Charles Hollerman, Dean, USD School of Medicine, would appreciate any "ideas" you might have concerning this concept. Thank you.

BCL,MD





The Strange Case Of The Double Pneumothorax

Ralph L. Read, M.D.*

1. A 46 year-old man developed a small left pneumothorax after placement of a left subclavian vein catheter. (Figure 1)
2. After a left chest tube was placed, supine AP (semi-upright) chest film showed successful re-

moval of the left pneumothorax. (Figure 2)
It also showed a linear density on the right which was thought to be another pneumothorax.
After a right chest tube was placed, repeat film showed persistence of the linear density, despite good positioning of the tube. (No figure)

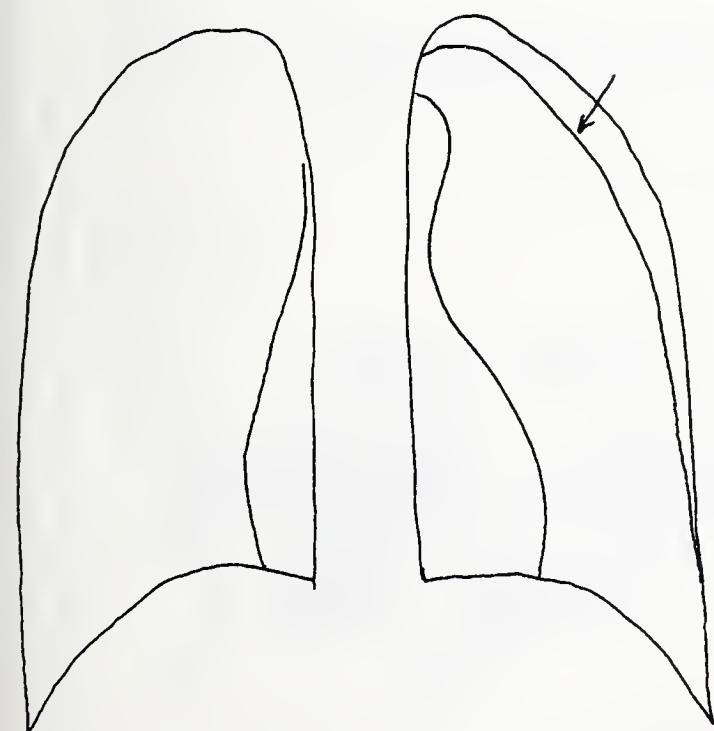


Figure 1

Portable A-P semi-upright chest X-ray. The visceral pleura is seen around the upper lung indicating pneumothorax.

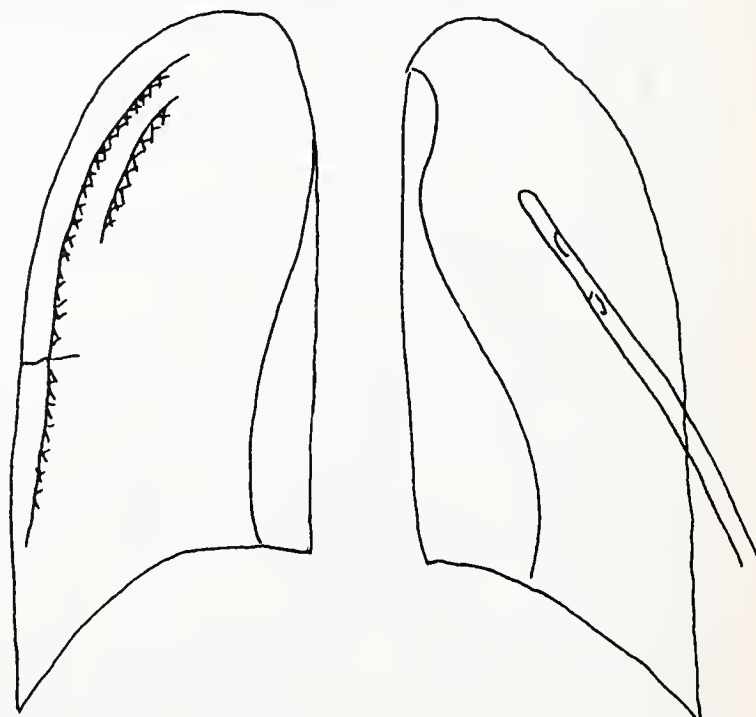


Figure 2

Portable A-P semi-upright chest X-ray. Chest tube on left has removed pneumothorax. On right side linear shadows are seen that resemble pneumothorax, but they persisted after placement of a right chest tube.

*Chief, Department of Radiology, University of South Dakota School of Medicine, Sioux Falls, South Dakota.

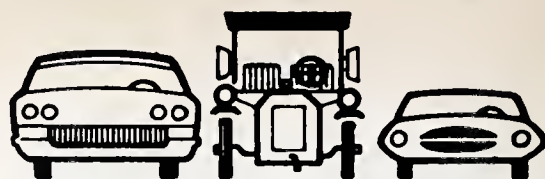
DIAGNOSIS: Pseudopneumothorax

EXPLANATION: Pseudopneumothorax is usually due to skin folds on the patients back. These skin folds may mimic the linear (pleural) shadow of pneumothorax, especially on semi-upright films because the patient "slides" down the film heaping up the skin on the back.

EXPLANATION: How to differentiate skin folds from pneumothorax.

1. There are vascular markings which pass "across" this linear shadow, which indicate the lung is not displaced from the parietal pleura. However, markings in the periphery are very small and may be difficult to see, especially on a dark film or an emphysematous patient.
2. If the interlobar fissure passes across the linear shadow, this shadow **cannot** be the edge of the lung.
3. The pleural line of pneumothorax is usually continuous over the highest part of the chest because of gravity. If a marginal line cannot be traced around the upper lung, suspect skin fold.
4. The marginal density of skin fold is produced by the heaping up of skin and is not a thin line as is the visceral pleura. If a marginal shadow has increased density along its inner aspect, suspect skin fold.
5. Skin folds are often multiple and bilateral. In this case, a smaller skin fold was present medial to the long one.

In difficult cases, a repeat film with efforts to eliminate skin folds can be the most reassuring proof!



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LETTER TO THE PROVIDERS IN SOUTH DAKOTA

Effective April 1, 1981, the South Dakota Foundation for Medical Care was awarded a one year grant by the Secretary of Health and Human Services to continue as the Professional Standards Review Organization for the State of South Dakota pursuant to Section 11.52(A) to review health care services and items provided in designated hospitals to persons eligible for benefits which may be paid for under the Medicare, Medicaid, and Maternal and Child Health and Crippled Children's Programs funded under Titles XVIII, XIX, and V of the Social Security Act. Individual files of providers will be located at South Dakota Foundation for Medical Care office at 608 West Avenue, North, Sioux Falls, South Dakota 57104.

Individual providers may have access to their individual information. The South Dakota Professional Standards Review Organization will provide health care practitioners and facilities, upon written request and payment of production or reproduction costs, copies of their individual PSRO data and information; permit such data to be corrected or amended where existing data is demonstrably incorrect; permit the physician of record or his designee, upon written request, to be present when a patient has access to his or her individual file.

The criteria utilized by the South Dakota Foundation for Medical Care in performing hospital reviews for Title XVIII, XIX, and V patients is the AMA screening criteria modified to reflect local practice patterns and the InterQual Intensity of Service/Severity of Illness Criteria. Effective August 1, 1981, the norms used in assigning length of stay checkpoints will be South Dakota length of stay norms, developed from actual length of stays of federally funded patients in South Dakota hospitals. When South Dakota length of stay norms cannot be developed (due to sample size), South Dakota Foundation for Medical Care will utilize the 1979 PAS North Central Region length of stay norms. Copies of criteria and length of stay norms utilized by South Dakota Foundation for Medical Care are available for inspection at 608 West Avenue, North, Sioux Falls, South Dakota.

For purposes of federal and state program monitoring, review and evaluation of privileged data and information may be only accessed by onsite visits to the PSRO or other components of the PSRO review system in which the privileged data and/or information is stored. All privileged information and data needed for monitoring and program review purposes must contain all personal identification in a coded form.

Non-privileged data and information acquired and/or generated by any PSRO, its agents, or ancillary components supporting PSRO review which is uniquely identifiable to a given health care facility may be disclosed upon request and payment of a fee to cover the expense of copying the requested information. However, the health care facility must be notified in writing 30 days prior to disclosure to permit the facility to review the information for accuracy and to provide comments to accompany the disclosed information.

Reports generated by the PSRO containing information required by federal agencies in their monitoring and program review capacity are considered to come under the Freedom of Information Act and once received by the Department of Health and Human Services are subject to its disclosure provisions.

Additional information may be obtained by contacting South Dakota Foundation for Medical Care, 608 West Avenue North, Sioux Falls, South Dakota 57104, Attention: L. Paul Jensen, Project Director.

THE ALUMNI ASSOCIATION

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1933 V. V. Rockey, M.D.	1962 W. N. Golliher, M.D.
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1948 C. H. Steele, M.D.	1979 Judith Gravdal, M.D.
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Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema, and bronchospastic reactivity to aspirin, iodides, or other non-steroidal anti-inflammatory agents. Anaphylactoid reactions have occurred in such patients.

Warnings: Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. *Motrin* should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If *Motrin* must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Chronic studies in rats and monkeys have shown mild renal toxicity characterized by papillary edema and necrosis. Renal papillary necrosis has rarely been shown in humans treated with *Motrin*.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue *Motrin* and the patient should have an ophthalmologic examination, including central visual fields and color vision testing. **Fluid retention and edema** have been associated with *Motrin*; use with caution in patients with a history of cardiac decompensation or hypertension. *Motrin* is excreted mainly by the kidneys. In patients with renal impairment, reduced dosage may be necessary. Prospective studies of *Motrin* safety in patients with chronic renal failure have not been done. *Motrin* can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy. Patients should report signs or symptoms of **gastrointestinal ulceration** or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema. To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged **corticosteroid therapy** should have therapy tapered slowly when *Motrin* is added. The antipyretic, anti-inflammatory activity of *Motrin* may mask inflammation and fever.

Drug interactions: *Aspirin*: used concomitantly may decrease *Motrin* blood levels.

Coumarin: bleeding has been reported in patients taking *Motrin* and coumarin.

Pregnancy and nursing mothers: *Motrin* should not be taken during pregnancy nor by nursing mothers.

Adverse Reactions

The most frequent type of adverse reaction occurring with *Motrin* is gastrointestinal, of which one or more occurred in 4% to 16% of the patients.

Incidence Greater Than 1% (but less than 3%)—Probable Causal Relationship

Gastrointestinal: Nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence); **Central Nervous System:** Dizziness,* headache, nervousness; **Dermatologic:** Rash* (including maculopapular type), pruritus; **Special Senses:** Tinnitus; **Metabolic/Endocrine:** Decreased appetite; **Cardiovascular:** Edema, fluid retention (generally responds promptly to drug discontinuation; see PRECAUTIONS).

Incidence Less Than 1%—Probable Causal Relationship**

Gastrointestinal: Gastric or duodenal ulcer with bleeding and/or perforation, gastrointestinal hemorrhage, melena, gastritis, hepatitis, jaundice, abnormal liver function tests; **Central Nervous System:** Depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma; **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome, alopecia; **Special Senses:** Hearing loss, amblyopia (blurred and/or diminished vision, scotomata, and/or changes in color vision) (see PRECAUTIONS); **Hematologic:** Neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs' positive), thrombocytopenia with or without purpura, eosinophilia, decreases in hemoglobin and hematocrit; **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure, palpitations; **Allergic:** Syndrome of abdominal pain, fever, chills, nausea and vomiting, anaphylaxis, bronchospasm (see CONTRAINDICATIONS); **Renal:** Acute renal failure in patients with preexisting, significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria; **Miscellaneous:** Dry eyes and mouth, gingival ulcer, rhinitis.

Incidence Less Than 1%—Causal Relationship Unknown**

Gastrointestinal: Pancreatitis; **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri; **Dermatologic:** Toxic epidermal necrolysis, photoallergic skin reactions; **Special Senses:** Conjunctivitis, diplopia, optic neuritis; **Hematologic:** Bleeding episodes (e.g., epistaxis, menorrhagia); **Metabolic/Endocrine:** Gynecomastia, hypoglycemic reaction; **Cardiovascular:** Arrhythmia (sinus tachycardia, sinus bradycardia); **Allergic:** Serum sickness, lupus erythematosus syndrome, Henoch-Schönlein vasculitis; **Renal:** Renal papillary necrosis.

*Reactions occurring in 3% to 9% of patients treated with *Motrin*. (Those reactions occurring in less than 3% of the patients are unmarked.)

**Reactions are classified under "Probable Causal Relationship" (PCR) if there has been one positive rechallenge or if three or more cases occur which might be causally related. Reactions are classified under "Causal Relationship Unknown" if seven or more events have been reported but the criteria for PCR have not been met.

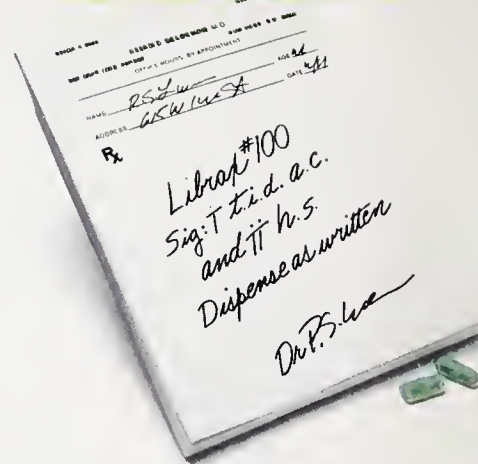
Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

Dosage and Administration: Do not exceed 2400 mg per day. If gastrointestinal complaints occur, administer with meals or milk.

Rheumatoid arthritis and osteoarthritis, including flares of chronic disease: Suggested dosage is 300, 400, or 600 mg t.i.d. or q.i.d. Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for relief of pain.

Caution: Federal law prohibits dispensing without prescription.

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Please consult complete prescribing information, a summary of which follows:

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Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction, hypersensitivity to chlordiazepoxide HCl and/or clidinium bromide.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage, withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression, suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants, causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated, avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction, changes in EEG patterns may appear during and after treatment, blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, i.e., dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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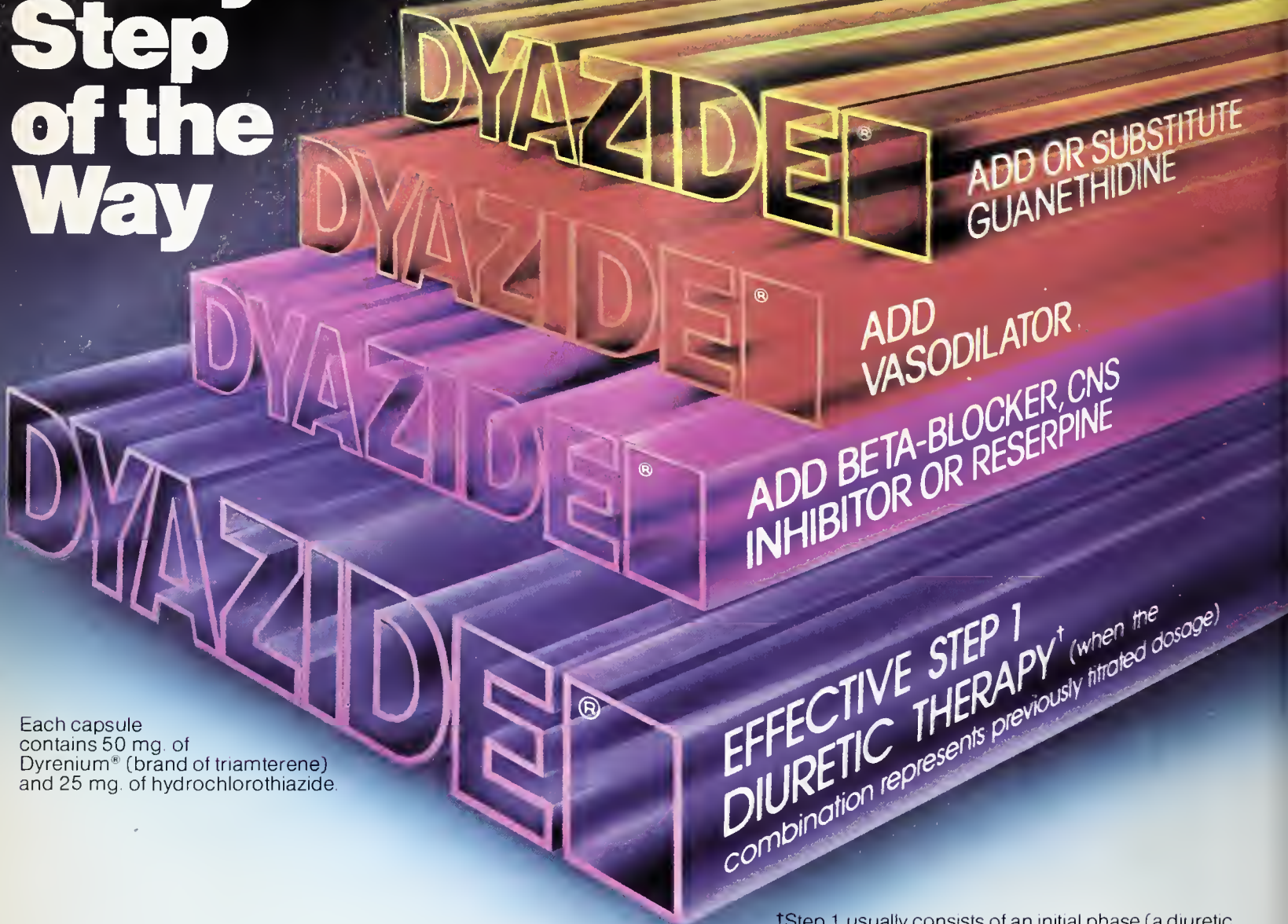
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WARNING

This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K⁺ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K⁺ intake. Associated widened QRS complex or arrhythmia requires prompt additional therapy. Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and

triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K⁺ frequently, both can cause K⁺ retention and elevated serum K⁺. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other, serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased

dietary intake of potassium-rich foods. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function.

Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth, anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis and of impotence have been reported with the use of 'Dyazide', although a causal relationship has not been established.

Supplied: Bottles of 1000 capsules; Single Unit Packages (unit-dose) of 100 (intended for institutional use only); in Patient-Pak™ unit-of-use bottles of 100.

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Fifty-Four Year Old Caucasian Male With Progressive Fatigability

G. Van Ert, M.D.*
J. F. Foss, M.D.**
Discussers

J. F. Barlow, M.D.***
Editor

Case No.: 705 182

This 54-year old Caucasian male was admitted to Sioux Valley Hospital in October, 1976 with chief complaint that for five months after an attack of shingles involving intercostal dermatomes 2 through 4 on the right, he had experienced increasing fatigability which markedly progressed particularly during the last month prior to admission and become associated with palpitations and shortness of breath on exertion over the last month. He had consulted a physician who had noted that he had a severe anemia with a hemoglobin of 5 gms/dl. A total serum protein of 9.7 gms/dl and electrophoresis interpreted as a monoclonal gammopathy were also detected.

There was no additional history on review of systems and, in particular, no weight loss or anorexia. He had no other hospitalizations or significant illnesses other than a fractured jaw 28 years prior to admission.

PHYSICAL EXAMINATION: Height: 6'1"; weight 160 lbs.; pulse 88/minute and regular; respiration 16/minute and regular; blood pressure 120 systolic and 80 diastolic. The patient was a well-developed, slender, fairly well-nourished, pale complexioned man who appeared chronically ill and fatigued. Examination of the head and neck revealed no palpable cervical adenopathy. A hard mass in the right lower neck was interpreted as the end of a cervical rib. There was a pytergyium of the right eye. The patient had a mild nosebleed at the time of physical examination. The lung fields were clear to auscultation and percussion. There was

a grade II soft blowing systolic murmur at the apex (VI grades). There was no tenderness, palpable organs or masses on abdominal examination. Rectal examination showed a slightly enlarged prostate which was not hard. Neurological examination was within normal limits.

LABORATORY DATA: Urinalysis, light yellow, clear, specific gravity 1.008, pH 6.5, negative for protein, glucose, ketone bodies, bile, and hemoglobin; sediment negative, hemoglobin 6 gms/dl, red count 1.51 million/mm³ ($1.51 \times 10^{12}/L$), hematocrit 15 vol/dl, mean corpuscular hemoglobin 31 micromicrograms, mean corpuscular volume 95 cubic micra (femtoliters), total leukocyte count 2,800/mm³ ($2.8 \times 10^9/L$), platelet count 62,000/mm³ ($62 \times 10^9/L$), reticulocyte count 2.7% uncorrected. Differential 50% segmented neutrophils, 1% neutrophilic bands, 46% lymphocytes, and 3% monocytes. There was marked rouleaux on the smear and mild hypochromasia. Lactic dehydrogenase (LDH), alkaline phosphatase, aspartate aminotransferase (SGOT), total bilirubin, calcium, direct bilirubin, serum iron, inorganic phosphorus, glucose, blood urea nitrogen, uric acid, cholesterol, sodium potassium and chloride were within normal limits. A creatinine was 1.4 mg/dl. Electrophoresis with a total protein 9.7 gms/dl revealed 3.4 gms/L albumin, 0.3 gms/L alpha I globulin, 0.8 gms/L alpha II globulin, 1.0 gms/L beta globulin, 4.2 gms/L gamma globulin with a monoclonal pattern on electrophoresis. Urine immunoelectrophoresis was negative. Serum IgG was 690 gms/dl (normal 800 to 1800 mgs/dl), IgM was 8100 mgs/dl, (normal 60 to 250 mgs/dl), IgA 42 mgs/dl (normal 90 to 450 mgs/dl). Electrocardiogram was read as borderline for left ventricular hypertrophy with ST-T wave changes most marked over the inferior lateral surface some of which may have been secondary to medications, ischemia, or left ventricular hypertrophy. A chest film was unremarkable. A skull film showed expansion of the sella turcica and tomography of this region was recommended. No lytic lesions were seen. A bone marrow examination was read as packed with immature lymphoid forms. The patient was transfused with 2 units of packed red cells which raised the hemoglobin to 7.2 gms/dl and was started on chlorambucil 10 mg/daily.

Eight weeks later the patient was readmitted because of anemia

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and was given four units of packed red cells raising the hemoglobin from 6.1 gms/dl to 11.0 gms/dl. A repeat bone marrow showed some decrease in the number of lymphocytes and plasma cell precursors and increase in normal elements. He was maintained on the same dosage of chlorambucil.

In 1979, a pterygium of the right eye was removed under local anesthesia. Hemoglobin was 13.6 gm/dl, white count $4800/\text{mm}^3$ ($4.8 \times 10^9/\text{L}$).

In 1980, he was admitted for increasing fatigability over the previous one and a half months and increased bruising. Hemoglobin was 9.2 gm/dl, white count $1300/\text{mm}^3$. He was given packed cell transfusions and discharged with a hemoglobin of 11 gm/dl. He was maintained on chlorambucil.

In late 1980, he was admitted with a hemoglobin of 7.0 gm/dl and a white count of $700/\text{mm}^3$ ($0.7 \times 10^9/\text{L}$). Platelets were $38,000/\text{mm}^3$ ($38 \times 10^9/\text{L}$). The bone marrow revealed 30% replacement by immature lymphoid forms. A cause for spiking fevers during hospitalization was not found. Blood, sputum, and urine cultures were negative. He was treated with gentamicin, carbenicillin and two packed red cell transfusions. He was kept on maintenance chlorambucil, 2 mgs daily. Three plasmaphereses of 3.5, 6.1 and 6.2 liter exchanges, were performed. One was performed during the last admission and two as out-patient procedures. The latest hemoglobin was 7.9 gm/dl, white count $1,500/\text{mm}^3$ ($1.5 \times 10^9/\text{L}$), platelet count $59,000/\text{mm}^3$ ($59 \times 10^9/\text{L}$). Total IgM had dropped to 1785 mg/dl.

DR. VAN ERT: This 54-year-old man presented with a chief complaint of increasing fatigability, dyspnea on exertion, and palpitations which progressed over a five month period after suffering an attack of shingles. A severe anemia, 5 gm/dl, was discovered in association with a total protein of 9.7 gm/L and an electrophoresis showing a monoclonal gammopathy. Past medical history was unremarkable and was significant for the absence of anorexia and weight loss. The initial differential diagnosis would include nonmyelomatous monoclonal immunoglobulinemia (NMMI), multiple myeloma (MM) and Waldenstrom's macroglobulinemia (WM), solitary plasmacytoma, malignant lymphoma and amyloidosis.

NMMI is eliminated quickly in view of the marked anemia. This entity is the most common disease state in which a monoclonal gammopathy is found but, by definition, must be present without evidence of malignant plasma cell dyscrasia. In NMMI the spike is usually less than 3 gm/L and plasma cells, which are normal, comprise less than 5% of the marrow. No lytic bone lesions and no anemia is demonstrated. Most importantly, there is no progressive abnormality in any of these tests over a period of time.

Physical examination in our patient showed no remarkable abnormality. In particular, there was no lymphadenopathy or hepatosplenomegaly. The findings, as presented in the protocol, would tend to exclude the diagnoses of malignant lymphoma or chronic lymphocytic leukemia in which lymphadenopathy or lymphocytosis in the peripheral smear should be seen.

The patient did have a nosebleed which is a common finding in WM due to interference of the mac-

roglobulin with clotting factors or platelet function. However, epistaxis is also seen with other abnormal monoclonal gammopathies as well as in MM. The lack of bone pain in the history is also significant and would not be expected in MM.

There was a normal urinalysis with no proteinuria. Since amyloidosis occurs in 15% of cases of MM and WM, deposition of the amyloid material in the kidney often gives rise to proteinuria. The amyloid is produced by the excretion of light chains from the abnormal clone of proliferating plasma-like cells in WM or MM. Although no gingival or rectal biopsy was done, secondary amyloidosis is unlikely in this case.

The patient had severe anemia but in patients with high levels of IgM, which is an intravascular protein (80%), increased plasma volume secondary to oncotic effects will often give rise to an accentuation of the anemia due to the increased plasma volume. Transfusions under these conditions can give rise to circulatory overload.

The findings of leukopenia and thrombocytopenia can be seen in WM. The reticulocyte count of 2.7% when corrected for the anemia easily drops to a normal or below normal values. The marked rouleaux formation seen in the peripheral blood is secondary to the abnormal serum protein.

Another significant finding in this case is not only the marked increase in IgM but the decrease in IgG and IgA. This is not infrequently seen in monoclonal gammopathy of either WM or MM. Especially significant in this case is bone marrow packed with immature lymphoid forms. This, in combination with the monoclonal IgM, the symptoms of progressive fatigue, dyspnea on exertion and bleeding tendency all point to a diagnosis of WM. It is amazing, considering the high levels of IgM, that there were not more findings of hyperviscosity which can often be indicated by segmentation of the retinal veins, with dilatation or hemorrhage. The electrocardiogram with left ventricular hypertrophy does suggest that the patient may have had a high plasma volume with high output failure. Lymphadenopathy and hepatosplenomegaly, which are common accompaniments of WM, were not present.

WM is almost exclusively a disease of the elderly with a peak incidence in the 6th and 7th decades. There is a male predominance and the average survival is 3-4 years.

In contrast, MM is generally characterized by severe bone pain with multiple lytic lesions of bone on X-ray. There is also a higher incidence of severe renal impairment in MM secondary to hypercalcemia with nephrocalcinosis, and a higher incidence of L-chain (Bence-Jones) proteinuria causing so-called myeloma kidney. Two-thirds of the mon-

clonal proteins in MM are IgG and another third are IgA. IgD and IgE are very rare. On screening electrophoresis 75% of patients with multiple myeloma will show a monoclonal spike. However, 10-15% of cases will show hypogammaglobulinemia with L-chain proteinuria only. In any case, the bone marrow examination shows greater than 10% plasma cells, many of which are atypical or immature. Hyperviscosity and bleeding tendency may also be seen in MM but less often than in WM. The average survival is about two years.

There is a problem in terminology as to what constitutes primary and secondary macroglobulinemia. Secondary macroglobulinemia is often seen in patients who otherwise have typical malignant lymphomas. There are also transitional or intermediate forms between typical WM and MM. Fudenberg and Vivella² recently have shown that the clear-cut distinctions are not always as clearcut as I have indicated. They described patients with monoclonal IgM but with typical lytic bone lesions characteristic of MM. They recommended that treatment be based upon clinical characteristics and course of the disease rather than on the protein class found.

Alkylating agents are the treatment of choice in WM. Objective responses include a decrease in the size of the liver and spleen, decrease in the serum viscosity and an increase in hemoglobin. Chlorambucil, given in a course with subsequent maintenance therapy, often leads to prompt response with relapse if therapy is discontinued. However, pancytopenia is a frequent consequence. Intermittent therapy with chlorambucil and prednisone has also been recommended. If this regimen fails, different drugs including adriamycin and bleomycin have been used.

Plasmapheresis has been effective adjunctive therapy since 80% of the IgM is intravascular. A decrease in the IgM may greatly relieve the hyperviscosity syndrome. Prognosis, however, seems to be related to the response to chemotherapy and pheresis does not seem to prolong survival.

**Dr. Van Ert's Diagnosis
Waldenstrom's Macroglobulinemia**

DR. FOSS: Laboratory evaluation plays an integral part both in the diagnosis and management of patients with dysproteinemias. Monoclonal or M proteins may be defined more specifically as immunoglobulins produced by cells of B lymphocyte derivation originating from a single clone of cells. Thus, the heavy and light chain components of immunoglobulin molecules produced are of identical amino acid composition. The laboratory can take advantage of this fact by performing serum protein electrophoresis. Since all proteins at a specific pH

carry differing amounts of electrical charge, depending on their amino acid arrangement, they may be separated one from the other by varying rates of migration when placed in an electrical field. If a monoclonal protein is present, it will appear as a distinct narrow band since it has an identical composition with a uniform electric charge.

In contrast, in infection, where a variety of different immunoglobulins have been produced, a broad or polyclonal band will be seen in the gamma region on electrophoresis.

According to Kyle, protein electrophoresis is the best test for screening patients for monoclonal gammopathy and should be used in all cases where MM, WM, or amyloidosis is suspected. As is demonstrated on the electrophoretic pattern in this example, a well demarcated M protein spike is present in the gamma region. (Fig 1).

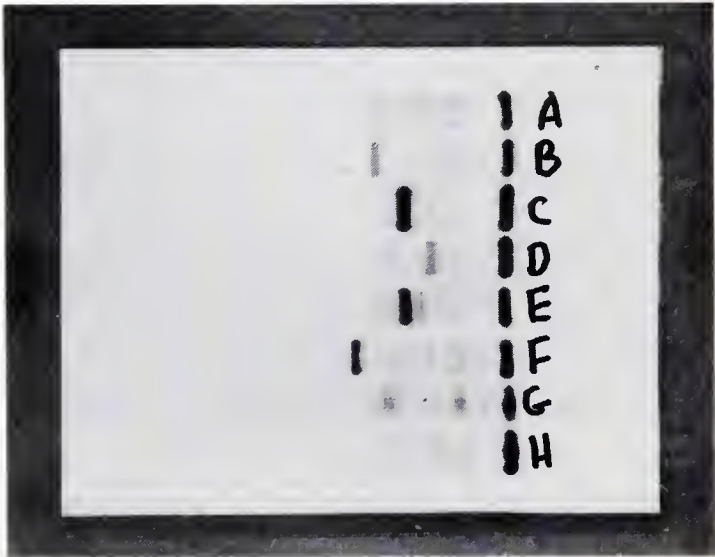


Figure 1
Serum protein electrophoretic patterns—A and H are normal patterns with albumin near designated letter at right. B-F show monoclonal like spikes ranging from beta to gamma region (furthest left). The pattern in G is normal but shows artifactual changes.

Once a monoclonal spike is identified on protein electrophoresis, it should be further characterized as to whether it is an artifact or real. If real, the specific immunoglobulin present should be determined, i.e. which heavy chain (IgG, IgA, IgM, IgD or IgE) and light chain (kappa or lambda) constitutes its composition. Serum immunoelectrophoresis is best designed to accomplish this purpose. This procedure consists of two steps. In the first the serum is subjected to an electrophoresis in a supporting gel similar to serum protein electrophoresis. Secondly, specific antisera for each of the heavy and light chains are placed in troughs cut parallel to the separated proteins. The antibodies are allowed to diffuse into the gel. As the antisera combine with

their corresponding antigens to form antigen antibody complexes, visible precipitin arcs are produced. The monoclonal protein, if present, will appear as a localized thickening or bowing of the precipitate line which can be contrasted with the normal-appearing precipitate lines. It must be remembered that this is a qualitative procedure. Only a subjective estimate of the amount of various immunoglobulins can be made i.e. greatly increased or greatly decreased. No actual quantitative levels can be obtained by immunoelectrophoresis. In Figure 2 one can see abnormal bowing of the IgM and kappa precipitin lines as compared to the normal.

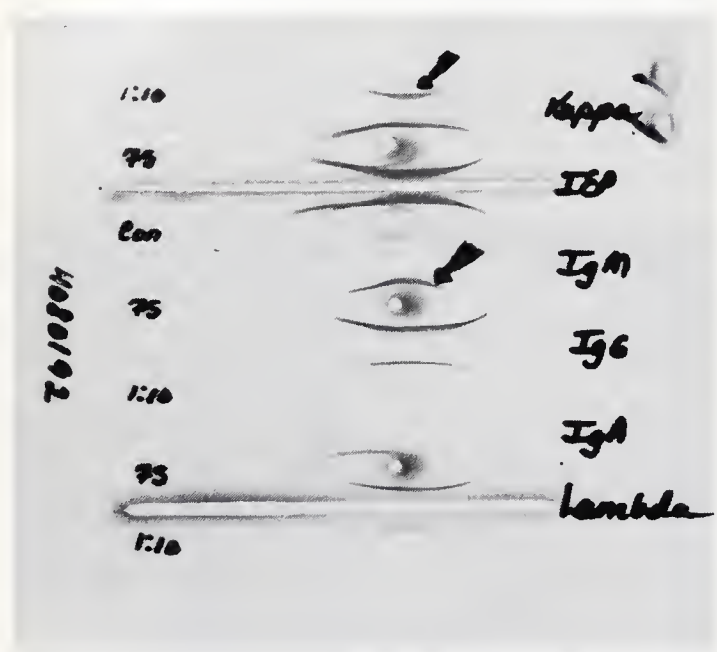


Figure 2

Serum protein Immunoelectrophoresis showing bowing and increased width of IgM and kappa indicating IgM kappa abnormality.

Analysis of urinary protein is also important in these patients to determine the presence or absence of monoclonal free light chains (Bence-Jones proteinuria). Unfortunately, the diagnostic "sticks" commonly used to detect protein in urinalysis are very insensitive to light chains, and, as such, should not be used for routine screening for Bence-Jones proteinuria. Indeed, urine immunoelectrophoresis or electrophoresis on concentrated urine are the methods of choice for detection of monoclonal protein in the urine.

If L-chains are present, a 24-hour urine protein determined by sulfosalicylic acid is the quantitative method that should be performed to monitor increases and decreases of the protein in the urine over time. This is a reflection of tumor burden. In this case no abnormal proteins could be identified in the urine, a common finding in WM.

Besides the above procedures, a quantitative method for measuring immunoglobulin called radial

immunodiffusion (RID) can be used. In this test, serum is placed within wells in an agar or agarose plate containing specific antiserum against the various heavy and light chains of the immunoglobulins. As the serum diffuses into the agar, a precipitate ring is formed when the zone of antigen-antibody equivalence is reached. By measuring the zone diameters of the precipitates and comparing these with zone diameters of known controls, actual serum immunoglobulin concentrations can be determined. This test is very reproducible when the particular immunoglobulin being measured is at very low or normal levels. However, with markedly increased levels of immunoglobulin, RID may be less reproducible depending upon the specific immunoglobulin in question. In the case of IgM, falsely elevated values occur frequently due to the presence of low molecular weight IgM moieties which migrate faster through the agar producing a larger zone diameter of precipitation. Less frequently, falsely low RID values may be encountered, probably due to high molecular weight IgM polymers. Therefore, a single method should be selected for quantitation of M protein when following a given patient. RID, if used consistently for a given patient, will reflect the correct relative changes of M protein levels resulting from therapeutic response or disease progression. Serum protein electrophoresis is probably the most reliable means of absolute protein quantitation for monitoring these patients.

In patients with monoclonal gammopathies, particularly of the IgM class, a frequent complication is hyperviscosity syndrome in which visual, central nervous system, or bleeding symptoms are present. In this case, it may be desirable to measure the serum viscosity. The viscosity or resistance to flow of the serum is usually compared to that of distilled water and expressed as a ratio. 0.8 to 1.0 is the upper limit of normal with symptoms of hyperviscosity occurring when the ratio becomes greater than 4:1.

A second problem encountered in approximately one-third of the patients with WM is the presence of cryoglobulins. Many patients with cryoglobulinemia will be asymptomatic but others will show such symptomatology as Raynaud's phenomenon, purpura, or cold urticaria. To test for cryoglobulins, the blood should be collected in a pre-warmed syringe and kept at 37°C. The serum is separated from the cellular portion of blood with half the serum being cooled to 4°C and the other half kept at 37°C for four hours. If cryoglobulins are present, either a clot or white precipitate will form in the refrigerated portion of the serum. The precipitate will disappear upon rewarming to 37°C. The height of the precipitate after centrifugation is a measure of amount of cryoglobulin and called the cryocrit.

In regard to the complications of bleeding, which are due to interaction of the abnormal protein with the clotting factors and platelets as well as manifestations of the hyperviscosity syndrome, one service the laboratory has to offer is plasmapheresis. Many times this procedure can result in marked amelioration of the patient's symptoms because the IgM molecule is large enough so that the majority is in the plasma compartment. A single five liter plasma exchange can remove as much as 80% of the abnormal immunoglobulin. This is of particular significance where emergency measures are required to quickly reduce immunoglobulin levels or alternatively in those patients who have had complications of chemotherapy or whose disease is not responding to chemotherapy.

In summary, laboratory procedures and tests play a central role in both the diagnosis and management of patients with dysproteinemias. With intelligent use they serve as useful adjunct to the clinician's clinical accumen. This case showed typical laboratory and morphologic findings.

FINAL DIAGNOSIS

WALDENSTROM'S MACROGLOBULINEMIA

*DR. BRADLEY RANDALL: It is important to remember that the monitoring of the therapeutic effect of plasmapheresis is difficult in regard to some cryoglobulins. The clinical response often does not correlate with laboratory tests. One test can be the use of the cryocrit in measurement of cryoglobulins. Unfortunately, the patient may not have as dramatic a clinical response as would be expected from the decrease in the cryocrit. This is probably because there is still abnormal cryoglobulin in the tissue which tends to reaccumulate in a period of time.

**DR. LOREN TSCHETTER: I would like to emphasize that one should screen for abnormal monoclonal proteins with the serum protein electrophoresis. It must be remembered that a considerable portion of patients will not have a monoclonal spike but will manifest hypogammaglobulinemia. In these cases there are often abnormal L chains in the urine. My preference is to obtain a 24-hour urine and measure a total protein as well as perform either a standard electrophoresis on concentrated urine or perhaps an immunoelectrophoresis. The total protein in the 24-hour urine and

serum protein electrophoresis can be used to monitor the patient. The immunoelectrophoresis on the serum is a qualitative test and is important but only has to be performed once to determine the specific abnormal protein.

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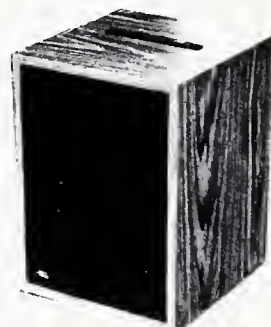
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- * Pathologist, Sioux Valley Hospital and Laboratory of Clinical Medicine, Sioux Falls, SD; Clinical Assistant Professor of Pathology in Laboratory Medicine, School of Medicine, The University of South Dakota.
- ** Specialist in Internal Medicine and Hematology, Central Plains Clinic, Sioux Falls, SD; Clinical Associate Professor of Medicine, School of Medicine, The University of South Dakota.

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Letters To The Editor

I was recently informed that I am one of the recipients of the SDSMA awards for 1981. I am indeed grateful and I would like to thank the Association for its generosity and recognition. This award provides me with more incentive to continue my study of medicine and hopefully provide quality health care to the people of South Dakota in the near future.

Sincerely,
John R. Fritz

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EFFECTIVE JULY 1, 1981

The article in the August 1981 Journal, "Toward a Middle Ground in the Technology Debate in Obstetric Care" was a reprint from *The Journal of Family Practice*, Vol. 12, No. 6: 971-972, 1981.

S D *Future Meetings*

September

The Disabled Worker: Overcoming the System's Barriers, Radisson South Hotel, Bloomington, MN, Sept. 21-22. Fee: \$150. 14 hrs. AMA Category I credits. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone: (612) 373-8012.

Third Adolescent Medicine and Health Care Conference, Focus on Adolescent Sexuality, Earle Brown Center, St. Paul Campus, St. Paul, MN, Sept. 23-24. Fee \$150—Physicians and \$80—Allied Health Prof. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455. Phone (612) 373-8012.

Fourth Annual Trauma Seminar, Hennepin County Medical Ctr., Minneapolis, MN, Sept. 24-26. Contact: CME, Box 293 Mayo Mem. Bldg., 420 Delaware St., SE, Minneapolis, MN 55455.

October

Current Issues in Perinatal Care, Holiday Inn of the Northern Black Hills, Spearfish, SD, Oct. 12-13. 9.6 hrs. CME credits. Contact: Margo Varcoc, R.N. Prog. Dir., SDPA, 1100 S. Euclid Ave., Sioux Falls, SD 57105. Phone: (605) 339-6578.

AAMI 1981 Regional Meetings, LA Marriott, Los Angeles, CA, Oct. 12-13. Fee: \$110 per course. Contact: Carl V. Hays, Prog. Coord., AAMI, 1901 N. Fort Myer Dr., #602, Arlington, VA 22209.

The 47th Annual Scientific Assembly of the American College of Chest Physicians, San Francisco Hilton Hotel & Civic Aud./Brooks Hall, San Francisco, CA, Oct. 25-29. 30 hrs. AMA Category I credits. Contact: Dept. of Education, AM. College of Chest Physicians, 911 Busse Highway, Park Ridge, IL 60068. Phone: (312) 698-2200.

November

AAMI 1981 Regional Meetings, Loews Anatole, Dallas, TX, Nov. 19-20. Fee: \$110 per course. Contact: Carl V. Hays, Prog. Coord., AAMI, 1901 N. Fort Myer Dr., #602, Arlington, VA 22209.

Diet and Exercise: Synergism in Health Maintenance, Walt Disney World Complex, Lake Buena Vista, FL, Nov. 3-4. AAFP and AMA Category I credits. Fee: \$60. Contact: Dept. of Foods & Nutrition, AMA, 535 N. Dearborn St., Chicago, IL 60610. Phone: (312) 751-6524.

Computer Tomography Scanning of the Brain, Masur Aud., NIH Clinical Ctr., Bldg. 10, Bethesda, MD, Nov. 4-6. Contact: Dr. Michael D. Walker, Dir., Stroke & Trauma Prog., Nat'l Institut. of Neuro. & Comm. Disorders & Stroke, Fed. Bldg., Rm. 8A08, 7550 Wisconsin Ave. Bethesda, MD 20205. Phone: (301) 496-2581.

AAMI 1981 Regional Meeting, Loews Anatole, Dallas, TX, Nov. 19-20. Fee: \$90 for full day, \$45 for evening & half day. Contact: Renee Pietranglo, AAMI, Suite 602, 1901 N. Ft. Myer Dr., Arlington, VA 22209. Phone: (703) 525-4890.

The Chemically Dependent Patient—A Physician's Update, Leamington Hotel, Minneapolis, MN, Nov. 14. Contact: Patricia Gurska, Dir. Public Rel., St. Mary's Hosp., 2414 S. Seventh St. Minneapolis, MN 55454. Phone: (612) 338-2229, ext. 406.

December

Behavioral Medicine and Primary Care in the '80's, Ilikai Hotel, Honolulu, HI, Dec. 4-11. 16 hrs. AAFP and AMA Category I credits. Fee: \$300. Contact: Jeri McClain, Adm. Assist., USC School of Med., Office of Academic Affairs, Columbia, SC 29208. Phone: (803) 777-7470.

American Cancer Society National Conference—Gastrointestinal Cancer—1981, Fontainebleau Hilton Hotel, Miami Beach, FL, Dec. 8-10. 13 hrs. AAFP & AMA Category I credits. Contact: Nicholas G. Bottiglieri, M.D., Am. Cancer Soc., 777 Third Ave., New York, NY 10017.

February

Rheumatology Seminar IV, South Seas Plantation Resort, Captiva Island, FL, Feb. 27—Mar. 6. 20 hrs. Category I credits. Fee: \$250. Contact Dept. of CME, MMA, Suite 400, Hlth. Assoc. Center, 2221 University Ave., SE, Minneapolis, MN 55414. Phone: (612) 378-1875.

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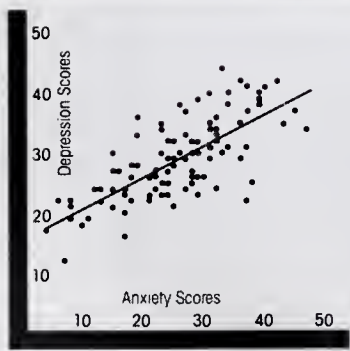
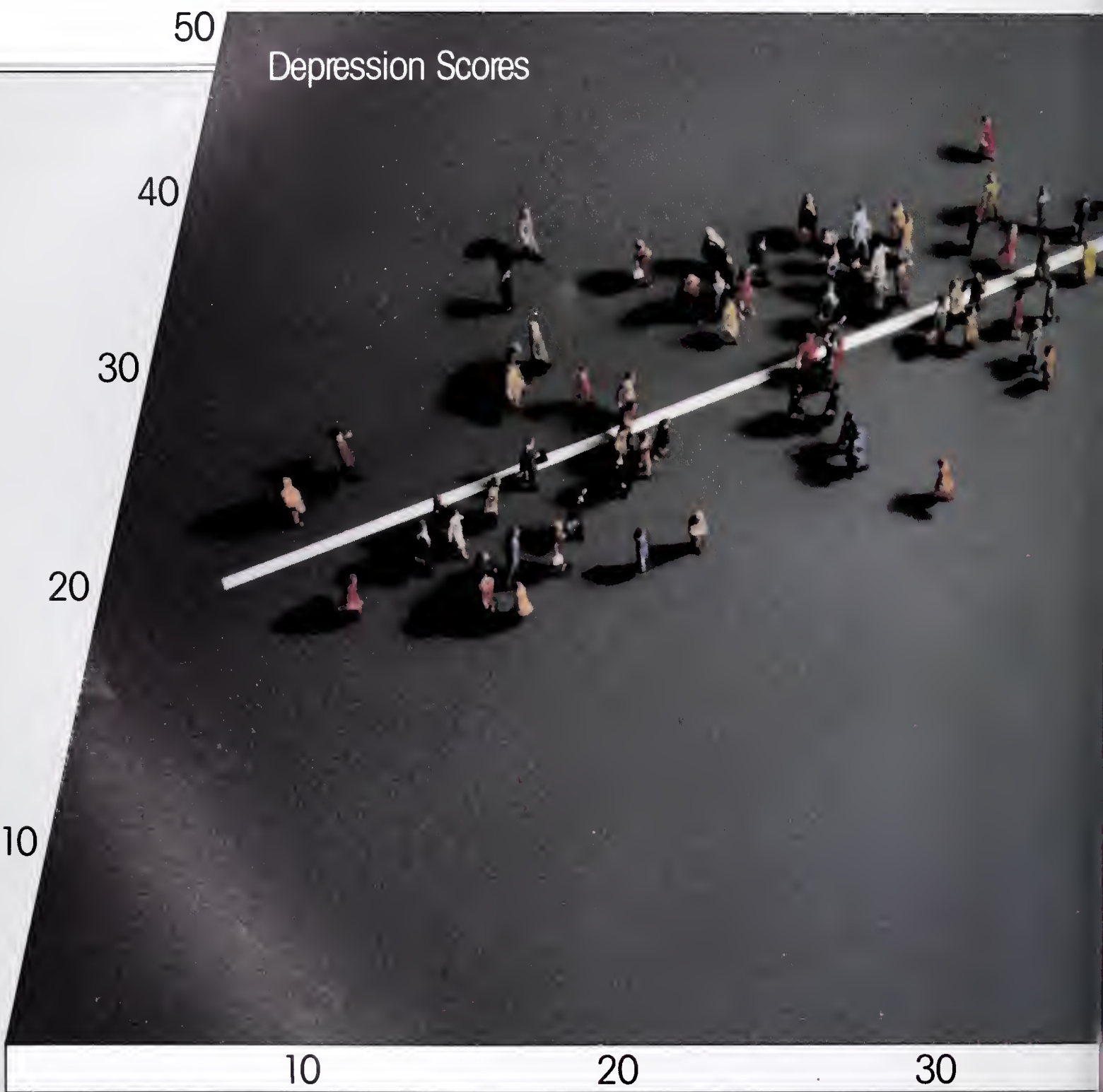
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FOR THE 7 OF 10 NONPSYCHOTIC



Clear correlation between anxiety and depression³

The above graph illustrates a relationship between anxiety and depression, indicating that patients seldom present with anxiety or depression alone; more often they have both in varying degrees. Data based on a sampling of 100 outpatients (64 male; 36 female) seen at a general psychiatric clinic.

³Adapted from Claghorn, J. The anxiety-depression syndrome. *Psychosomatics* 11:438-441, Sept-Oct 1970.

DEPRESSED PATIENTS WHO ARE ALSO ANXIOUS^{1,2}

Most depressed patients are also anxious. . .

Some authors estimate that 70% of all nonpsychotic patients with symptoms of depression have concomitant symptoms of anxiety.^{1,2} One author found a distinct correlation between anxiety and depression scores in 100 nonpsychotic outpatients administered the Minnesota Multiphasic Personality Inventory in a general psychiatric clinic.³ As depression scores increased, so did anxiety scores. No attempt was made to select patients other than to exclude psychotics.

but not psychotic

The logic of treating both components of anxious depression is clear. Antipsychotics, like the phenothiazines, however, carry a well-documented risk of tardive dyskinesia.⁴ Because of this, an APA Task Force recently recommended the judicious use of phenothiazines in cases other than chronic psychosis or the use of alternative treatments.

A better way to give relief

Limbitrol combines the specific anxiolytic action of Librium® (chlordiazepoxide HCl/Roche)—a benzodiazepine with a long history of safe use—with the antidepressant action of amitriptyline, a tricyclic of established clinical efficacy. In comparison to phenothiazines, Limbitrol and its components have rarely been associated with tardive dyskinesia or other extrapyramidal side effects. And in terms of rapid response and patient compliance, Limbitrol appears to be superior to amitriptyline alone. Controlled multiclinic studies showed Limbitrol relieved more symptoms more rapidly than did amitriptyline.⁵ Despite a higher incidence of drowsiness, the dropout rate due to side effects was lower with Limbitrol. (See adverse reactions section in summary of product information on next page. As with any CNS-acting agent, patients should be cautioned about driving or using dangerous machines while on therapy with Limbitrol.)

References: 1. Rickels K: Drug treatment of anxiety, in *Psychopharmacology in the Practice of Medicine*, ed. Jarvik ME. New York, Appleton-Century-Crafts, 1977, p. 316. 2. Schatzberg AF, Cole JO: Benzodiazepines in depressive disorders. *Arch Gen Psychiatry* 35:1359-1365, 1978. 3. Claghorn J: The anxiety-depression syndrome. *Psychosomatics* 11:438-441, 1970. 4. The Task Force on Late Neurological Effects of Antipsychotic Drugs: Tardive dyskinesia, summary of a task force report of the American Psychiatric Association. *Am J Psychiatry* 137:1163-1172, 1980. 5. Feighner JP et al: A placebo-controlled multicenter trial of Limbitrol versus its components (amitriptyline and chlordiazepoxide) in the symptomatic treatment of depressive illness. *Psychopharmacology* 61:217-225, 1979.

Anxiety Scores

50

In moderate depression and anxiety

Limbitrol[®] IV

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

Relief without a phenothiazine

Please see summary of product information on next page.

LIMBITROL® TABLETS Tranquilizer—Antidepressant

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of moderate to severe depression associated with moderate to severe anxiety

Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use, then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.

Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.) Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving).

Usage in Pregnancy: Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage, withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline, symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).

Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated; sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12.

In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.

Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.

The following list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:

Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.

Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.

Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.

Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.

Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.

Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.

Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single *h.s.* dose may suffice for some patients. Lower dosages are recommended for the elderly. Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.

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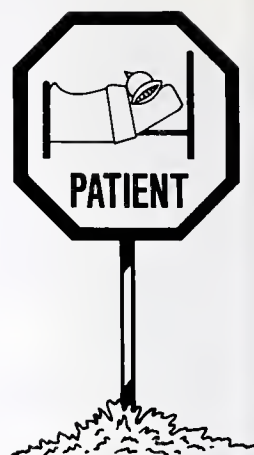
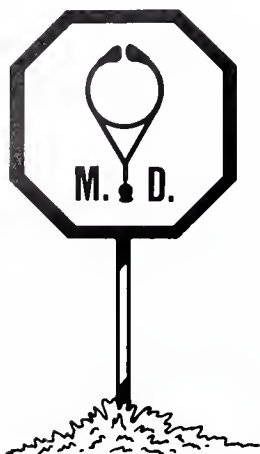
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Clinicopathological Conference
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With Right Upper Quadrant Pain



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Malignant Hyperthermia: A Case Report

Donald H. Knudson, M.D.*
Michael Rost, M.D.**
LuAnn Grann, C.R.N.A.***

ABSTRACT

Malignant hyperthermia is a pharmacogenetic muscle disorder which every surgeon, anesthesiologist and anesthetist should be prepared to diagnose and treat promptly. This phenomenon can be initiated by inhalation anesthesia, local anesthetics, or muscle relaxants. It is inherited through an autosomal dominant gene and occurs most often among young, healthy, muscular patients.

Malignant hyperthermia occurs at the rate of approximately one per 10,000 patients given general anesthesia. Reported mortality rates have usually

been greater than 50%. It occurs most frequently with general anesthesia, but can also be triggered by local and regional anesthetics. Many of the patients affected by this process have previously been noted to have neuromuscular defects such as hernias, squints, and scoliosis or kyphosis. A familial history of death by an unknown cause and occurring while the family member was under anesthesia should alert the physician to explore the possibility of this genetically-transmitted condition.

The following case report is presented as a representative example of this disorder.

Case Report

J. R. is a nine year old 25 kg. male who developed abdominal pain on the morning of admission. He was admitted to McKennan Hospital from the emergency room. He had had a history of recurrent intussusceptions, with surgical reductions at age 13 months and six years. Admission vital signs were temperature 101° F, pulse 104, respirations 24, blood pressure 84/64. Chest film was negative. Laboratory data showed a white blood count of 9,600, hemoglobin 14.2, hematocrit 40.2. Electrolytes were normal.

The patient's condition did not change overnight, and he presented to the operating room on the following day for an exploratory laparotomy with a preoperative diagnosis of recurrent intussusception

versus intestinal obstruction from other causes. Pre-operative anesthetic interview was essentially negative. The patient's father denied any previous anesthetic problems in the patient's family or in the patient's previous operations. The previous operations had been at a different hospital so no old records were available.

On arrival in the operating room, the patient was sleepy from his preoperative medication of Demerol 25 mg. and Atropine 0.3 mg. The patient was pre-treated with 1.5 mg. curare and preoxygenated for five minutes with 100% oxygen per mask. Vital signs at the time of induction were pulse 80, respirations 24, blood pressure 140/70. Due to the possible bowel obstruction, a rapid 150 sodium pentothal induction with the reverse trendelenburg and cricoid pressure was performed. 40 mg. of succinylcholine was given as a relaxant. During an initial attempt at intubation, the jaw was not adequately relaxed so an additional 10 mg. of succinylcholine was given intravenously. Adequate relaxation was obtained after about 30

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seconds. No muscle rigidity was noted at the time other than the poorly relaxed mandible, which, after additional succinylcholine, did relax. After intubation, the vital signs were blood pressure 110/60, pulse 120, respirations were controlled. An esophageal temperature probe was inserted, and initial reading showed 100.5 degrees F. Anesthesia was maintained for the duration of the case with halothane .5-1.5%, nitrous oxide 50% and oxygen 50%. Findings at operation were dense adhesions, causing an obstruction near the ileocecal valve. This was thought to be secondary to previous surgery. No intussusception was noted. Due to the intense degree of adhesions and scarring, a short segment of bowel was resected and an end-to-end anastomosis performed. During the operation, the patient's vital signs were normal. When the peritoneal cavity was opened and part of the intestine placed outside the abdomen for inspection, the patient's temperature began to drift downward, with the lowest temperature recorded at 98.7 degrees F. An additional 20 cc. of .2% succinylcholine drip was used during closure of the abdominal cavity. At this time, his temperature had slowly risen to 99.3 degrees F. Halothane was discontinued beginning with the closure of the abdomen. Following closure of the abdomen, the patient began breathing spontaneously, coinciding with cessation of the succinylcholine drip. Respirations were 56 per minute and sinus tachycardia increased. Bilateral breath sounds were equal and his skin color was normal. Following closure of the skin, the patient was placed on 100% oxygen. Mouth was suctioned and an esophageal temperature probe was removed and a rectal probe was inserted. Temperature was 100° F, blood pressure 110/56, pulse 140, respirations 56. During the next five minutes, the patient first developed cyanotic nail beds and then became rigid with his entire hands and lower arms turning cyanotic. Leg mottling was noted. Tachycardia rose to 150 per minute, and temperature to 101 degrees F. At this point, 25 mg. of dantrolene was pushed intravenously, and the patient was packed in ice. A Foley catheter and nasogastric tube were inserted, and sterile iced saline was lavaged. Because of the patient's marked muscular rigidity, blood gases were unable to be drawn. 25 meq of sodium bicarbonate was given intravenously. The patient's temperature continued to rise steadily to 103.8 degrees F. and sinus tachycardia to 160 per minute. No cardiac arrhythmias were noted.

10 mg. of Lasix intravenously was given 30 minutes following the onset of hyperthermia. The patient's temperature remained at 103.8 degrees. An additional 15 mg. of dantrolene was given intravenously. His hands remained cyanotic. An addi-

tional 50 meq of sodium bicarbonate was given intravenously. Vital signs at this time showed a pulse of 140 and respirations of 40. Over the next ten minutes the patient's rigidity began to lessen, and the patient responded to verbal commands. An arterial line was established and blood gases were drawn while the patient was breathing 100% oxygen. Blood gases showed a pH of 7.51, pCO₂ 34, HCO₂ 27, base excess +6, pO₂ 499.

40 minutes following onset of hyperthermia, the patient's temperature had decreased to 102 degrees F. 20 additional mg. of dantrolene was given intravenously. The iced lavage was discontinued and the ice was removed from around the patient's body. 50 minutes following the onset of hyperthermia the patient was alert and temperature was 100 degrees F. He was suctioned, extubated, and moved to the Intensive Care Unit. His hands remained cyanotic but leg mottling was decreased.

The patient remained in the Intensive Care Unit for 48 hours. Hypothermia blanket was used during the first few hours to keep his temperature below 102 degrees rectally. His hands remained cyanotic, but color gradually improved with warm packs to the hands.

The patient had a satisfactory postoperative course. He remained afebrile in the acute care unit and also throughout his postoperative hospital course. He was discharged from the hospital one week following operation in satisfactory condition.

Clinical Manifestations

Although hyperthermia can be initiated by general or local anesthetics, the classical combination noted to cause the syndrome has been halothane in combination with succinylcholine. Fever, skeletal muscle rigidity, arrhythmias, hyperpnea, cyanosis, arterial oxygen desaturation, and respiratory and metabolic acidosis.

Serum CPK may be elevated in members of families susceptible to this disease. However, normal enzyme levels of this enzyme do not exclude the possibility of the occurrence of this syndrome.

The earliest sign is often increased muscle tone after administration of succinylchloride. Tightness of the jaw in these patients prior to insertion of the endotracheal tube has frequently been noted by anesthesia personnel. Cardiac arrhythmias, especially PVCs and tachycardia, usually occur prior to temperature elevation. Fever usually begins to occur within 30 to 60 minutes following induction of anesthesia. Mortality is directly related to the severity of the fever. The core body temperature has been noted to rise by as much as 2° C every five minutes, until death ensues.

Simultaneous with the temperature rise, the pa-

tient usually develops respiratory acidosis. Often hypocalcemia and hypokalemia develop, while serum phosphorus and CPK rise. If the process is not arrested, muscle necrosis is inevitable, with resulting myoglobinuria, renal failure, electrolyte imbalance, hypermetabolism, and brain death.

Treatment

The single most important factor in reducing mortality from malignant hyperthermia is early recognition and detection of the problem. Careful monitoring of the patient's body temperature throughout the operative procedure is imperative if the syndrome is to be treated in its early stages.

Once malignant hyperthermia is suspected, the operation should be concluded as expeditiously as possible, concomitant with cessation of anesthesia. The patient should be administered 100% oxygen to combat the greatly increased metabolic rate and to prevent hypoxia. Intravenous sodium bicarbonate (NaHCO_3) and mechanical hyperventilation are indicated for prevention of the marked respiratory and metabolic acidosis which inevitably occurs. Diuretics, such as mannitol and furosemides, should be given to clear myoglobin and other muscle catabolism products, in order to prevent renal failure.

Rapid cooling is mandatory, if survival is to be achieved. The patient should be packed in ice and given iced saline through nasogastric and bladder catheter tubes.

Although the exact etiology of malignant hyperthermia is unclear, it is thought that failure of the muscle sarcoplasmic reticulum to take up calcium is an important mechanism. This apparently results in recurrent contraction of the sarcomeres and resultant muscle rigidity and eventual catabolism. In August of 1979, an intravenous preparation of dantrolene sodium (Dantrium) became available. It has been in use as a muscle relaxant for several years. This drug specifically reduces the outflow of calcium from the sarcoplasmic reticulum, although the precise mechanism of action is unknown.

The use of dantrolene has proved to be invaluable in lowering the morbidity and mortality of this syndrome. It should be given intravenously as soon as malignant hyperthermia is suspected, in the dosage of 1 mg/kg. Repeat doses can be given every 10 to 15 minutes to a total of 10 mg/kg, for short term use. Side effects have been negligible. Oral dantrolene is also available for prophylactic use in patients suspected of having this disease.

Summary

Malignant hyperthermia is an autosomal dominant muscular disease capable of causing sudden death in the operating room. Early diagnosis and

treatment of the condition is essential, and every surgeon, anesthesiologist, and anesthesiologist should be prepared to treat it. The recent introduction of intravenous dantrolene into the medical armamentarium has offered promise of lowering the current high mortality rate.

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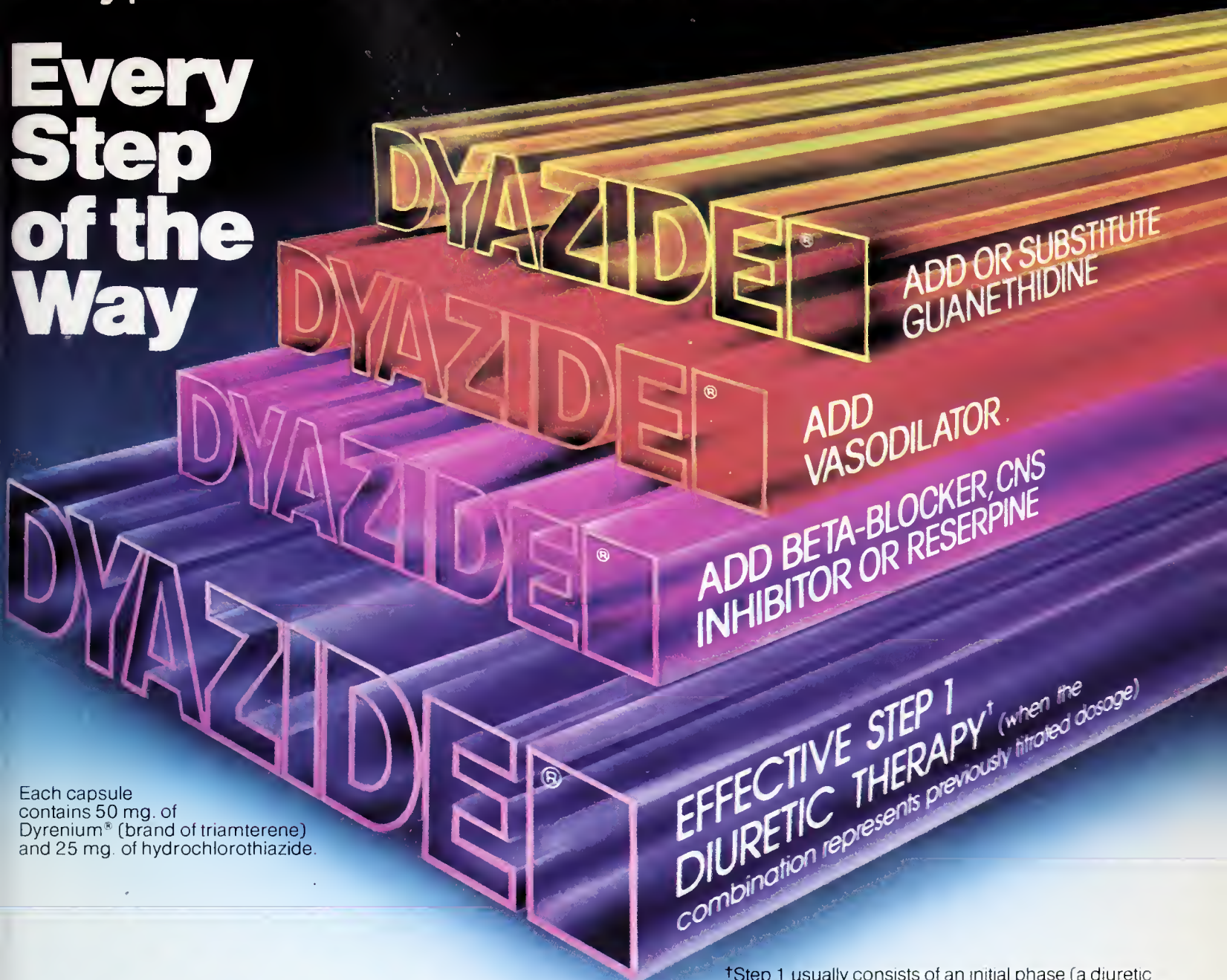
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triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K⁺ frequently, both can cause K⁺ retention and elevated serum K⁺. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded; in the other, serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased

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Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth, anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and rarely, allergic pneumonitis have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis and of impotence have been reported with the use of 'Dyazide', although a causal relationship has not been established.

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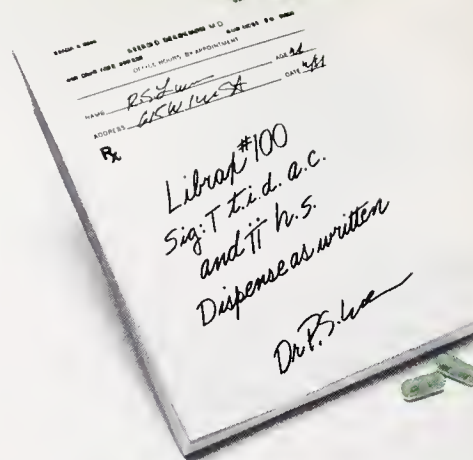


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Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially, increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression, suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated, avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction, changes in EEG patterns may appear during and after treatment, blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, i.e., dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.



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Irritable BOWEL SYNDROME*

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A visible difference in myoelectric rhythms of the colon

Studies reveal an increased frequency of 3-cycles-per-minute slow wave basic electrical activity in the colons of patients with IBS—a significant difference in basic colonic rhythm patterns from normal subjects.^{1,2} These findings suggest a physiological basis for the spasm and hypermotility characteristic of IBS. The role of severe anxiety in triggering or aggravating such symptoms has long been recognized. Consequently, treatment should focus on both aspects of the problem.

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References: 1. Sullivan MA, Cohen S, Snape WJ. *N Engl J Med* 298:878-883, Apr 20, 1978.
2. Snape WJ et al: *Gastroenterology* 72: 383-387, Mar 1977.

Specify **Librax**®
Adjunctive

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium Br.

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*Librax has been evaluated as possibly effective for this indication. Please see summary of prescribing information on facing page.

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An effective short-term adjunct in an indicated weight loss program

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In uncomplicated obesity

Many patients, on the other hand, present with excess fat but no disease. While this condition is often termed uncomplicated obesity, complications of both a social and a psychologic nature may be distressingly real for the patients. In these cases, a short-term regimen of Tenuate can help reinforce your dietary counsel during the important early weeks of an indicated weight loss program.

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The anorectic effectiveness of diethylpropion hydrochloride is well documented. No less than 18 separate double-blind, placebo-controlled studies attest to its usefulness in daily practice.¹ And the unique chemistry of Tenuate provides "... anorectic potency with minimal overt central nervous system or cardiovascular stimulation."² Compared with the amphetamines, diethylpropion has minimal potential for abuse.

Tenuate—it makes sense.
And it's responsible medicine.

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References: 1 Citations available on request from Merrell Dow Pharmaceuticals Inc., Cincinnati, Ohio 45215. 2. Hoekenga M T et al: A comprehensive review of diethylpropion hydrochloride. In Central Mechanisms of Anorectic Drugs, S. Garattini and R. Samanin, Ed., New York. Raven Press, 1978, pp. 391-404.

Tenuate[®] 
(diethylpropion hydrochloride USP)
Tenuate Dospan[®] 
(diethylpropion hydrochloride USP)
controlled-release
AVAILABLE ONLY ON PRESCRIPTION

Brief Summary
INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.
CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).
WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. When central nervous system active agents are used, consideration must always be given to the possibility of adverse interactions with alcohol. *Drug Dependence:* Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy:* Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children:* Tenuate is not recommended for use in children under 12 years of age.
PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.
ADVERSE REACTIONS: *Cardiovascular:* Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System:* Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal:* Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic:* Urticaria, rash, ecchymosis, erythema. *Endocrine:* Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System:* Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous:* A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.
DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg tablet three times daily, one hour before meals, and in mid evening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.
OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine[®]) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of June, 1980
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Merrell Dow

Child Passenger Protection

In normal every-day wear and tear, children hold up very well. But in a car that stops suddenly, or crashes, many children don't do well at all.

Why? Because so many parents don't realize how dangerous an automobile is to young children. And they haven't done anything to protect their children.

Imagine a car traveling at just 30 miles per hour. That is the speed the passengers are traveling too. Then there is a crash . . . and the car stops but the passengers keep moving at 30 miles per hour until they are all stopped by something . . . with the force of a fall from a three-story building. That can be rough on someone, especially a child. (A ten-pound child becomes a 300 pound projectile in a car going 30 miles per hour.)

Maybe you can understand how automobile crashes are the number one killer of children . . . ahead of all other types of accidents and diseases. They are also a major cause of epilepsy and paraplegia.

But there is an easy cure for this epidemic: child restraints and safety belts. Thus far, in 1981, five states have either enacted a "child-passenger protection law" for the first time or amended an existing law. Generally, the term "child-passenger protection law" refers to state legislation mandating that children below a specified age (usually 3 or 4) be properly protected through the use of child-passenger restraint systems meeting certain standards, when being transported in motor vehicles upon the streets and highways of the state.

Arguments for passage of such legislation include the following:

1. Motor vehicle accidents account for the leading cause of death and serious injury to all children beyond infancy up to the age of 39.
2. Over 90 percent of the children ride unprotected.
3. Child restraint devices have been shown to reduce the chances of death by more than 90 percent and serious injury by 80 percent.
4. Educational efforts to convince adults to wear seat belts have been unsuccessful.
5. National Highway Transportation Safety Administration estimates each death costs \$240,000 and \$7,000 for each injury. Thus the proposed law represents a practical approach toward health-cost containment.
6. Mandatory seat belt use is required in many other industrial nations of the world. (23 in all.)

Tennessee passed the initial Child Restraint legislation in the United States in 1977 with the help of Doctor Robert S. Sanders, a pediatrician from

Murfreesboro, Tennessee, and with the endorsement of the Tennessee Medical Association and the specialty societies of the state. Minnesota passed its legislation this year, effective the first of January 1982. Rhode Island, Illinois and West Virginia are the other states with existing legislation.

"Individual Liberties" got the motor cycle helmet laws repealed in many states in past years. Most doctors and experienced cyclists know helmets should be worn—law or no law. The same argument of freedom of choice has made child restraint legislation unpopular. We should consider that small children have no say in deciding if they will take the greatest risk of their short lives.

Statistics have indicated that 940 children under the age of 5 died in motor vehicle accidents in 1976 and that same year, some 57,000 children were injured, which is over 160 per day. Parents buckle up their babies and children less than they do themselves. Studies agree that only 6-7 percent of the children are being restrained in the cars while 18 percent of the adults wear seat belts.

The South Dakota State Medical Association is on record supporting public education programs concerning "Child Passenger Protection". With that in mind, what can we as doctors do, to increase the voluntary use of child restraints and then safety belts in South Dakota? Some of the suggestions made by the office of Highway Safety planning in Lansing, Michigan are as follows:

1. Obtain educational materials from sources such as the American Academy of Pediatrics.
2. Consider having an infant or toddler seat on display in your office. (often these are available from automobile dealers)
3. Educate your office personnel to restraint importance.
4. Compare restraints with immunizations, and stress the importance of both. (Motor vehicle accident risk is higher!)
5. Write a prescription for a safe car seat for any child who doesn't have one.
6. Encourage community service groups to expand availability of child car seats at low cost by developing rental programs in your communities. (Childbirth Education Classes have often undertaken this as a project.)
7. Conduct an educational program on auto safety for the doctors in your area.

For printed and audio-visual materials, contact either the National Highway Traffic Safety Administration in Washington, D.C. or the Office of Highway Safety Planning, Lansing, Michigan 48913.



Bruce Lushbough, M.D., President
South Dakota State Medical Association

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(meprobamate and ethoheptazine citrate with aspirin) Wyeth

Twofold analgesic action teamed with time-proven efficacy against concurrent anxiety and tension in patients with musculoskeletal disease.*

EQUAGESIC—Abbreviated Summary

***INDICATIONS:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective for the treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease or tension headache.

Final classification of the less-than-effective indications requires further investigation.

The effectiveness of Equagesic in long-term use, i.e. more than four months, has not been assessed by systematic clinical studies. The physician should periodically reassess usefulness of the drug for the individual patient.

CONTRAINDICATIONS: Equagesic should not be given to individuals with a history of sensitivity or severe intolerance to aspirin, meprobamate, or ethoheptazine citrate.

WARNINGS: Careful supervision of dose and amounts prescribed for patients is advised, especially with those patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g., alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on or habituation to the drug. Where excessive dosage has continued for weeks or months, dosage should be reduced gradually rather than abruptly stopped, since withdrawal of a "crutch" may precipitate withdrawal reaction of greater proportions than that for which the drug was originally prescribed. Abrupt discontinuance of doses in excess of the recommended dose has resulted in some cases in the occurrence of epileptiform seizures. Special care should be taken to warn patients taking meprobamate that tolerance to alcohol may be lowered with resultant slowing of reaction time and impairment of judgment and coordination.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with the use

of minor tranquilizers (meprobamate, chlordiazepoxide, and diazepam) during the first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physicians about the desirability of discontinuing the drug. Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breast-feeding patients, the drug's higher concentration in breast milk as compared to maternal plasma levels should be considered.

Preparations containing aspirin should be kept out of the reach of children. Equagesic is not recommended for patients 12 years of age and under.

PRECAUTIONS: Should drowsiness, ataxia, or visual disturbance occur, the dose should be reduced. If symptoms continue, patients should not operate a motor vehicle or any dangerous machinery.

Suicidal attempts with meprobamate have resulted in coma, shock, vasomotor and respiratory collapse, and anuria. Very few suicidal attempts were fatal, although some patients ingested very large amounts of the drug (20 to 40 gm). These doses are much greater than recommended. The drug should be given cautiously, and in small amounts, to patients who have suicidal tendencies. In cases where excessive doses have been taken, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Hyperventilation has been reported occasionally. Any drug remaining in the stomach should be removed and symptomatic treatment given. Should respiration become very shallow and slow, CNS stimulants, e.g., caffeine, Metrazol, or amphet-

amine, may be cautiously administered. If severe hypotension develops, pressor amines should be used parenterally to restore blood pressure to normal levels.

ADVERSE REACTIONS: A small percentage of patients may experience nausea with or without vomiting and epigastric distress. Dizziness occurs rarely when meprobamate and ethoheptazine citrate with aspirin is administered in recommended dosage. The meprobamate may cause drowsiness but, as a rule, this disappears as therapy is continued. Should drowsiness persist and be associated with ataxia, this symptom can usually be controlled by decreasing the dose, but occasionally it may be desirable to administer central stimulants such as amphetamine or mephentermine sulfate concomitantly to control drowsiness.

A clearly related side effect to the administration of meprobamate is the rare occurrence of allergic or idiosyncratic reactions. This response develops, as a rule, in patients who have had only 1-4 doses of meprobamate and have not had a previous contact with the drug. Previous history of allergy may or may not be related to the incidence of reactions.

Mild reactions are characterized by an itchy urticarial or erythematous, maculopapular rash which may be generalized or confined to the groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema, and fever have also been reported.

More severe cases, observed only very rarely, may also have other allergic responses, including fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case), and hyperthermia. Treatment should be symptomatic such as administration of epinephrine, antihistamine, and possibly hydrocortisone. Meprobamate should be stopped, and reinstitution of therapy should not be attempted.

Rare cases have been reported where patients receiving meprobamate suffered from aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia. In nearly every instance reported, other toxic agents known to have caused these conditions have been associated with meprobamate. A few cases of leukopenia during

continuous administration of meprobamate are reported; most of these returned to normal without discontinuation of the drug.

Impairment of accommodation and visual acuity has been reported rarely.

OVERDOSE: Two instances of accidental or intentional significant overdosage with ethoheptazine citrate combined with aspirin have been reported. These were accompanied by symptoms of CNS depression, including drowsiness and light-headedness, with uneven recovery. However, on the basis of pharmacological data, it may be anticipated that CNS stimulation could occur. Other anticipated symptoms would include nausea and vomiting. Appropriate therapy of signs and symptoms as they appear is the only recommendation possible at this time. Overdosage with ethoheptazine combined with aspirin would probably produce the usual symptoms and signs of salicylate intoxication. Observation and treatment should include induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions.

DESCRIPTION: Each Equagesic tablet contains 150 mg meprobamate, 75 mg ethoheptazine citrate and 250 mg aspirin.

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*This drug has been evaluated as possibly effective for this indication.

Wyeth Laboratories
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Down with pain

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for mild to moderate pain

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More than twice as much acetaminophen as the leading combination plus a full therapeutic dose of propoxyphene...all in a convenient, economical single tablet.

WYGESIC—Abbreviated Summary

INDICATION: For the relief of mild-to-moderate pain.

CONTRAINDICATION: Hypersensitivity to propoxyphene or to acetaminophen.

WARNINGS: CNS ADDITIVE EFFECTS AND OVER-

DOSAGE: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics or other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. Toxic effects and fatalities have occurred following overdoses of propoxyphene alone or in combination with other CNS depressants. Most of these patients had histories of emotional disturbances or suicidal ideation or attempts, as well as misuse of tranquilizers, alcohol, or other CNS-active drugs. Caution should be exercised in prescribing large amounts of propoxyphene for such patients (see **Management of Overdosage**).

DRUG DEPENDENCE: Propoxyphene can produce drug dependence characterized by psychic dependence and less frequently physical dependence and tolerance. It will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics. The abuse liability of propoxyphene is qualitatively similar to codeine's although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine.

USAGE IN AMBULATORY PATIENTS: Propoxyphene may impair the mental and/or physical abilities required for potentially hazardous tasks, e.g. driving a car or operating machinery. Patients should be cautioned accordingly.

USAGE IN PREGNANCY: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. **INSTANCES OF WITHDRAWAL SYMPTOMS IN THE NEONATE HAVE BEEN REPORTED FOLLOWING USAGE DURING PREGNANCY.** Therefore, propoxyphene should not be used in pregnant women unless, in the

judgement of the physician, the potential benefits outweigh the possible hazards.

USAGE IN CHILDREN: Propoxyphene is not recommended for children because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric group.

PRECAUTIONS: Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine. The CNS depressant effect of propoxyphene may be additive with other CNS depressants, including alcohol.

ADVERSE REACTIONS: The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting. These seem more prominent in ambulatory than in nonambulatory patients; some of these reactions may be alleviated if the patient lies down.

Other adverse reactions include constipation, abdominal pain, skin rashes, light-headedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances. The chronic ingestion of propoxyphene in doses over 800 mg per day has caused toxic psychoses and convulsions. Cases of liver dysfunction have been reported.

DRUG INTERACTIONS: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, and other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended (see **Warnings**). Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine.

MANAGEMENT OF OVERDOSAGE: SYMPTOMS: The manifestations of serious overdosage with propoxyphene are similar to those of narcotic overdosage and include respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, cyanosis), extreme somnolence progressing to stupor or coma, pupillary constriction, and circulatory collapse. In addition to these characteristics which are reversed by narcotic antago-

nists such as naloxone, there may be other effects. Overdoses of propoxyphene can cause delay of cardiac conduction as well as focal or generalized convulsions, a prominent feature in most cases of severe poisoning. Cardiac arrhythmias and pulmonary edema have occasionally been reported, and apnea, cardiac arrest, and death have occurred.

Symptoms of massive overdosage with acetaminophen may include nausea, vomiting, anorexia, and abdominal pain, beginning shortly after ingestion and lasting for 12 to 24 hours. However, early recognition may be difficult since early symptoms may be mild and nonspecific. Evidence of liver damage is usually delayed. After the initial symptoms, the patient may feel less ill, however, laboratory determinations are likely to show a rapid rise in liver enzymes and bilirubin. In case of serious hepatotoxicity, jaundice, coagulation defects, hypoglycemia, encephalopathy, coma, and death may follow. Renal failure due to tubular necrosis, and myocardiopathy have also been reported.

Ingestion of 10 grams or more of acetaminophen may produce hepatotoxicity. A 13-gram dose has reportedly been fatal.

TREATMENT: Primary attention should be given to the reestablishment of adequate respiratory exchange through provision of a patent airway and institution of assisted or controlled ventilation. The narcotic antagonists, naloxone, nalorphine, and levallorphan are specific antidotes against the respiratory depression produced by propoxyphene. An appropriate dose of one of these antagonists should be administered preferably I.V., simultaneously with efforts at respiratory resuscitation and the antagonist should be repeated as necessary until the patient's condition remains satisfactory. In addition to a narcotic antagonist, the patient may require careful titration with an anticonvulsant to control seizures. Analeptic drugs (e.g. caffeine or amphetamine) should not be used because of their tendency to precipitate convulsions.

Oxygen, IV fluids, vasopressors and other supportive measures should be used as indicated. Gastric lavage may be helpful. Activated charcoal can absorb a significant amount of ingested propoxyphene. Dialysis is of little value in poisoning by propoxyphene alone. Acetaminophen is rapidly absorbed, and efforts to remove the drug from the body should not be delayed. Copious gastric lavage and/or induction of emesis may be indicated. Activated charcoal is probably ineffective unless administered almost immediately after acetaminophen ingestion. Neither forced diuresis nor hemodialysis appears to be effective in removing acetaminophen. Since acetaminophen in overdose may have an antidiuretic effect and may produce renal damage, administration of fluids should be carefully monitored to avoid overload. It has been reported that mercaptamine (cysteine) or other thiol compounds may protect against liver damage if given soon after overdosage (8-10 hours). N-acetylcysteine is under investigation as a less toxic alternative to mercaptamine, which may cause anorexia, nausea, vomiting, and drowsiness. Appropriate literature should be consulted for further information (JAMA 237:2406-2407, 1977). Clinical and laboratory evidence of hepatotoxicity may be delayed up to one week. Acetaminophen plasma levels and half-life may be useful in assessing the likelihood of hepatotoxicity. Serial hepatic enzyme determinations are also recommended.

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Wyeth Laboratories
Philadelphia, PA 19101



Fifteen Month Old Female Referred For Abnormal Chest Film

Michael Justice, M.D.*
Discusser

John F. Barlow, M.D.**
Editor

Case No. 885 312-9

This 15-month-old caucasian toddler female was admitted to Sioux Valley Hospital with chief complaint of abnormal chest X-ray and difficulty in maintaining gait.

The patient had been well until three to four weeks prior to admission when she was admitted to a Winner hospital for pneumonia. Initial chest X-ray revealed a cystic mass in the left lower lobe and a significant shift of the heart and the mediastinum to the right. The patient required oxygen and steam during admission. The pneumonia cleared but the cystic mass persisted. She was referred to Yankton where she had a bronchoscopy which revealed no foreign body in the bronchus. Because the cystic mass seemed to be enlarging, she was referred in for further evaluation. The child did seem to be having some difficulty breathing and increasing instability of the legs in the days prior to admission.

The patient was involved in a motor vehicle accident at 5-½ months of age but apparently was not knocked unconscious and no definite injury was sustained. The mother had had a normal gestation with a vaginal delivery. Birth weight was 8 lbs. There was some hyperbilirubinemia during the postpartum period but no other abnormality. The patient was breast fed. There was no history of contact with tuberculosis or other known diseases.

PHYSICAL EXAMINATION: Temperature 98.6°F; pulse 100/min. and regular; respirations 44/min. and regular; height and weight were appropriate for age; blood pressure was 128 systolic and 70 diastolic. There were no abnormalities of the head and neck except for a slightly bulging left ear drum. The neck was supple. Examination of the chest showed diminished breath sounds in the left lower lung field and hyperresonance to percussion. The heart was pushed to the right but had a regular sinus rhythm

without murmurs. There were no palpable organs, masses, or tenderness in the abdomen. The genitalia were normal female. The child was slightly bow-legged, and had a broad based gait but no other abnormality. The neurologic examination was within normal limits.

LABORATORY DATA: Urinalysis yellow, clear; specific gravity 1.011; pH 6.0, negative for protein, glucose, ketone bodies, bile and hemoglobin; sediment—negative. Hemoglobin 12.6 gm/dl, hematocrit 30 Vol/dl with normal blood indices. Total leukocyte count 11,100/mm³ (11.1 x 10⁹/L) with 39% segmented neutrophils, 2% neutrophilic bands, and 59% lymphocytes. Platelet count was 139,000/mm³ (139 x 10⁹/L). The red cells were normochromic, normocytic on smear. A lactic dehydrogenase (LDH) was 332 IU/L (normal 0-270 IU/L), alkaline phosphatase, total protein, calcium, phosphorus, glucose, blood urea nitrogen, creatinine, uric acid and electrolytes were within normal limits for age. A chest film showed a cystic lesion at the left base with a shift of the mediastinum and heart to the right. An operation was performed.

DR. JUSTICE: The differential diagnosis in this case is essentially one of a cystic lung lesion in a 15-month-old child. This type of lesion could be either congenital or acquired. Any type of lung lesion causing localized obstruction of a bronchus with secondary localized emphysema could present this type of picture. Examples would include endobronchial foreign body, endobronchial tuberculosis, tuberculosis of the tracheobronchial lymph nodes, and endobronchial or mediastinal tumors. The diagnosis of these diseases would be supported by fluoroscopy when, during expiration, the emphysematous area does not decrease in size. Therefore, I believe that the most likely etiology for this child's cystic lung lesion is congenital.

* Resident in Family and Community Medicine, Sioux Falls, SD.

** Medical Director of Laboratory, Sioux Valley Hospital and pathologist, Laboratory of Clinical Medicine, Sioux Falls, SD; Professor of Pathology and Laboratory Medicine, School of Medicine, The University of South Dakota.

Classifying the lesion into an acquired or congenital category is difficult. According to Ravitch and Hardy the ultimate criterion for this distinction is the gross and microscopic picture. A cystic lesion of the lung is considered congenital if a similar pathologic picture can be found in the prenatal or immediate neonatal period, prior to any postnatal traumatic or inflammatory insult, or if anomalous blood supply is present at the time of diagnosis.

There are 4 major categories of congenital cystic disease of the lung. They are: 1) lobar emphysema—This is a postnatal overdistension of one or more lobes of a histologically normal lung. This is thought to result from cartilagenous deficiency in the tracheobronchial tree; 2) cystic adenomatoid malformation—This is a multicystic mass of pulmonary tissue in which there is a proliferation of bronchial structures at the expense of alveoli. The disease is considered a focal pulmonary dysplasia and the cysts are lined by cuboidal or columnar epithelium; 3) pulmonary sequestration—This is a cystic mass of nonfunctioning pulmonary tissue that lacks normal communication with the tracheobronchial tree and which receives most or all of its arterial blood supply from anomalous vessels; and 4) bronchogenic cyst—This is a discrete mass of nonfunctioning pulmonary tissue which usually has no communication with the tracheobronchial tree.

Buntain et al⁹, at a Children's hospital in Los Angeles reviewed 64 cases of congenital cystic lesions of the lung from 1947 to 1972. The following characteristics were found. There was a 1.3 to 1.0 ratio of males to females. There was no ethnic or racial predominance and no familial occurrence. One third of the patients in whom a diagnosis was made in the neonatal period and more than half of the patients were detected before 6 months of age. Patients with lobar emphysema and cystic adenomatoid malformation were noted early and bronchogenic cysts and pulmonary sequestration were found later in life. Right and left-sided involvement was nearly equal. Except for pulmonary sequestration, there was upper and middle lobe preponderance. No patient in their series had bilateral involvement. Fifty-nine percent of the patients presented with respiratory distress, 10% had recurrent infections and 11% were asymptomatic. Other presentations included feeding difficulties, persistent cough, or chest pain.

Thirty-seven percent of the patients had other associated anomalies. Chief among these were intracardiac defects which were associated mostly with lobar emphysema. The second most common anomaly was pectus excavatum. There were no

anomalies in the patients with bronchogenic cysts.

I believe that our patient has one of these congenital cystic lung lesions. Lobar emphysema is a possibility. However, this usually involves an upper lobe or the right middle lobe of the lung. The respiratory symptoms caused are generally severe and occur shortly after birth but may be delayed for several months. The chest film is compatible with our case showing a lobar involvement with shift of the mediastinum. Treatment for this disease is surgical excision and prognosis is very good. The factors against this diagnosis in our case are the late age of onset of symptoms and the location of the cystic lesion.

Bronchogenic cysts occur close to the midline and become symptomatic when they become infected or when they enlarge in size and compress the surrounding lung. Fever and productive cough are the most common persisting complaints. Treatment for symptomatic cysts is surgery and antibiotics. Asymptomatic cysts may be followed. The main factor against this being the diagnosis in our case is the position of the cyst since bronchogenic cysts occur more often centrally.

Pulmonary sequestration is usually asymptomatic until infection occurs. The sequestration usually occurs in the lower lobes and presents during childhood, most commonly with recurrent pneumonia. Bronchography shows a mass of intrathoracic tissue without connection to the airways. Aortography confirms the diagnosis by demonstrating that it receives its blood supply by an anomalous aortic artery. There may be a continuous or purely systolic murmur heard over the back. Treatment is surgical excision and antibiotics for infection. Asymptomatic sequestrations should also be removed because of the high incidence of infection.

The other possibility is cystic adenomatoid malformation. Our patient's chest film and physical examination would be compatible with this disease. However, the involvement is more commonly in an upper lobe. The majority of patients become symptomatic and die in the newborn period, but other patients may be asymptomatic until mid-childhood. The latter group of patients present with respiratory disease or recurrent infections. Treatment is surgical excision.

Dr. Justice's Diagnosis

Congenital Cystic Adenomatoid Disease Of The Lung, Possible Pulmonary Sequestration

*DR JAMES REYNOLDS: I would like to point out on the chest film the marked shift of the mediastinum and heart to the right. If you don't orient the film properly you can occasionally miss this finding. (Fig. 1)

* Thoracic Surgeon, Sioux Valley Hospital, Sioux Falls, South Dakota.

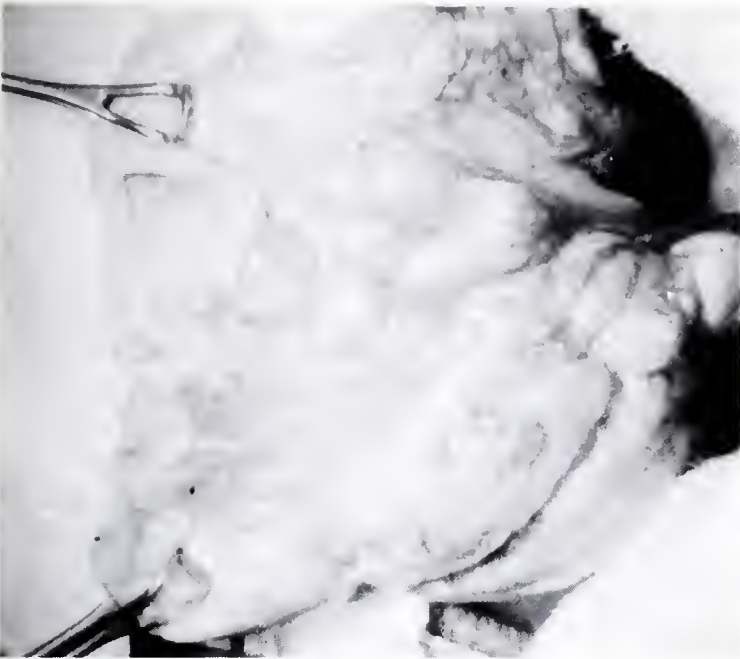


Figure 1

Chest X-ray revealing radiolucency at left base and shift of the heart into the right chest.

There was one important piece of information that was not given in the protocol. An aortogram was performed and this failed to show any systemic blood supply to the lesion in the left lower lobe. This would essentially exclude a diagnosis of pulmonary sequestration. I might also add one more lesion to the differential diagnosis of cystic lesions of the left lower lobe. This would be a diaphragmatic hernia. These lesions can persist and not be detected until long after the neonatal period. However, I believe a gastrointestinal series was performed when the patient was in Yankton. Therefore, this diagnostic possibility was excluded.

I am not always sure you need an aortogram to determine whether there is a systemic circulation to the lung as one sees in pulmonary sequestration, as long as the surgeon realizes that pulmonary sequestration with a systemic blood supply is possible.

We resected the left lower lobe. This is the gross picture of the cystic lung at the time for surgery. (Fig. 2) Because of the normal aortogram, our pre-operative diagnosis was congenital cystic adenomatoid malformation of the lung. One interesting observation during surgery was an anomalous vascular pattern about the hilus of the lung. The child has done extremely well postoperatively and has had no complications.

DR. BARLOW: Submitted was the left lower lobe which was enlarged and had multiple blebs on the pleural surface. Cut section revealed a multicystic mass with focal mucus retention. Microscopically, the cystic spaces were lined by ciliated respiratory epithelium with a small amount of smooth muscle and elastic tissue in the walls. Normal alveoli are easily discerned between the cystic spaces. (Fig. 3)



Figure 2

Gross specimen at surgery revealing multiple cysts.

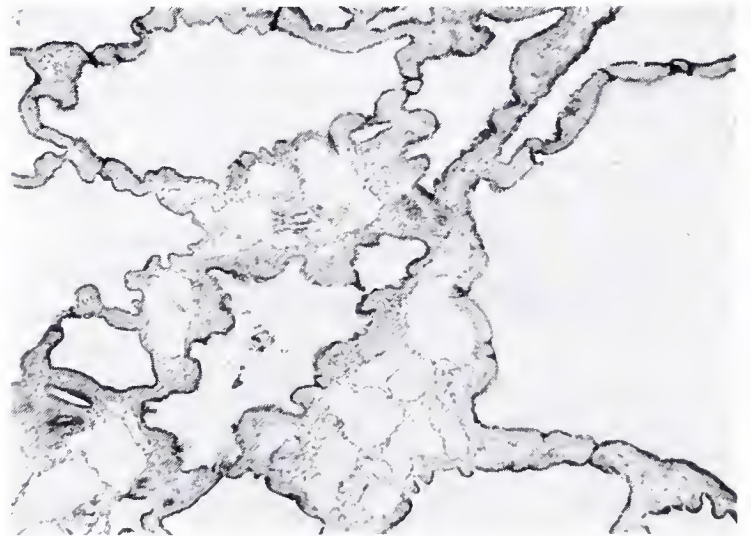


Figure 3

Microscopic of lung showing multiple cysts lined by respiratory epithelium with intervening normal alveoli.

FINAL ANATOMIC DIAGNOSIS CONGENITAL CYSTIC ADENOMATOID MALFORMATION OF THE LUNG

This entity has a variety of synonyms—cystic adenomatoid malformation of the lung, congenital cystic lung disease, cystic fetal bronchial adenoma, adenomatoid hamartoma, cystic hamartoma, and congenital bronchiolar malformation.

It was first described by Chin and Tang in 1949,^{6,7} although Stork^{6,7} had described the entity in 1897 in Germany. In 1978, Ostor and Fortune³ reviewed 142 cases of this rare entity.

The lesion is usually unilateral and limited to a lobe but markedly expands the lobe causing compression of surrounding lung and a shift of the mediastinum. The lesion has been reported with no constant predilection for sex or area of lung involved.

Pathologically, it has been defined by a variety of morphologic criteria. Grossly, the lesion may be solid, microcystic or multicystic corresponding to the classification of Van Dijk and Wagenvoort⁸ (cystic, intermediate, or solid). Occasionally one large cyst dominates the picture. Microscopically, the following are stated criteria: 1) absence of cartilage, 2) absence of bronchial glands, 3) foci of columnar mucinous metaplasia, 4) overproduction of terminal bronchiolar structures without alveoli except subpleurally (modified), 5) massive enlargement of affected lobe, 6) papillary infolding of mucosa with increased elastic tissue in supporting cyst walls, and 7) lack of an inflammatory component—usually rejected for pneumatocele if inflammation is present. The type of epithelium lining the cysts is ciliated or non-ciliated pseudostratified or cuboidal except as above.

There have been a variety of pathologic and clinicopathologic subclassifications. Bale² divided the lesions into two basic types. One was a solid radioopaque mass with or without cystic areas with marked bronchiolar epithelial proliferation, immature or no alveoli and mucinous epithelium. This was seen in stillborns or prematures with anasarca or hydrops fetalis and was associated with maternal hydramnios. The other major type was predominantly cystic with milder bronchiolar epithelial proliferation and occasional areas of mucinous epithelium and was seen in term or older infants with no anasarca and was not associated with maternal hydramnios. The major clinical presentation was respiratory distress early in life. Our case represents this latter form.

Stocker et al^{6,7} in a large series described three types as follows: A) Type I—single or multiple cysts lined by respiratory epithelium associated with smooth muscle and elastic tissue in the walls. The cysts are often large or a single cyst can be dominant. Cartilage is rare. Mucinous epithelium is seen in a third of the cases and normal alveoli are present between the cysts. The usual clinical presentation is severe respiratory distress in early infancy; B) Type II—multiple more uniform cysts lined by cuboidal or columnar respiratory bronchiolar epithelium. There is striated muscle in the walls in some cases but no mucinous epithelial lining or cartilage in the walls. Alveoli are present between the cysts. There is clinical respiratory distress but there is a high incidence of associated severe congenital anomalies including cystic renal disease, renal age-

nesis or dysgenesis with Potter's facies or syndrome, intestinal atresia, cardiac anomalies, cleft palate, hydraencephaly, hydrocephalus and diaphragmatic hernia; C) Type III—large bulky noncystic lesion producing mediastinal shift with glandular structures lined by cuboidal epithelium with or without cilia.

I believe our case fits a type I of Stocker. The presence of rare cartilage and smooth muscle but no striated muscle in the wall suggest Type I pathologically. However, there was more uniformity to the cyst size than a classic Type I. The unilateral lobar enlargement with compression of the surrounding lung and imperceptible blending of normal and abnormal lung fit well with Type I. The radiologic picture and clinical presentation are also compatible with Type I. The absence of associated congenital anomalies is also helpful.

This entity may be present clinically in three ways: 1) in a stillborn or neonatal death with nonimmunologic hydrops fetalis or anasarca associated with material hydramnios, 2) progressive neonatal respiratory distress with cyanosis, dyspnea, tachypnea, and subcostal retraction. Diminished breath sounds over the area is frequent. This is a surgical emergency due to shift of the mediastinum and compression of the surrounding lung by the large unilateral lobar mass; and 3) recurrent pulmonary infection is the least common presentation. Tension pneumothorax may complicate the condition.

There has been considerable discussion over the pathogenesis of the disorder. It is most likely a developmental defect at the bronchiolar level of the lung stemming from an insult from the third to tenth week of fetal life. Some have suggested the defect must be between the lobar differentiation of the lung at the fifth week but before cartilage development at the tenth week of gestation. It has been suggested that the hydramnios is due to lack of absorption of fluid by the compressed normal lung.

Diagnosis is usually made by chest roentgenogram but the disease has also been diagnosed prenatally by ultrasound.

When the disease is present as respiratory distress, surgical lobectomy must be performed. An anomalous blood supply can create surgical difficulty.

I might also like to say a word about the differential diagnosis. By definition pulmonary sequestration indicates there is no connection with the bronchial tree and a systemic arterial blood supply above or below the diaphragm is present. There are two types of pulmonary sequestration. One is extrapulmonary sequestration which occurs as an extrapulmonary mass often near the left lower lobe. It is frequently symptomatic in the neonatal period and is associated with a variety of congenital anomalies. The second type, intrapulmonary sequestra-

tion, is usually only noted later in childhood when infection supervenes.

Another entity is bronchopulmonary foregut malformation which indicates a portion of pulmonary parenchyma with a connection to the gut with or without a systemic blood supply.

Congenital lobar emphysema has already been well discussed.

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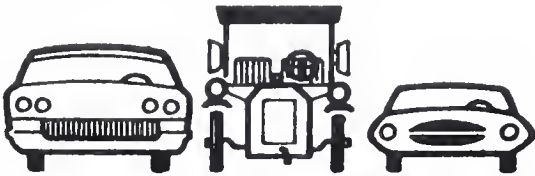
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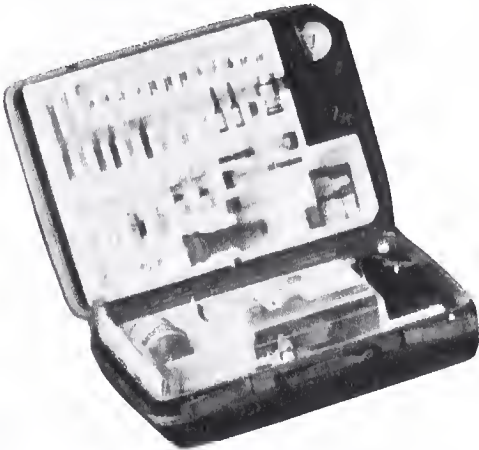
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Centennial President Honored

Bruce C. Lushbough, M.D. of Brookings, Centennial President of the SDSMA, was honored by SDAFP at their noon luncheon during the Centennial Convention in Sioux Falls. Dr. Lushbough, President of SDAFP 1973-74, was presented with a plaque, commemorating his installation as Centennial President, by SDAFP President R.G. Nemer of Gregory.

AAFP Request Project Proposals

The AAFP Project on research in Health Care Delivery now administered by the Academy's Committee on Research, is funded with \$15,000 for the 1981-82 fiscal year. The committee will accept applications for funding or partial funding of research projects directly related to health care delivery.

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Principles of Colon and Rectal Surgery, Mayo Mem. Aud., U. of Minn., Minneapolis, Minn., Oct. 21-24. 26 hrs. AMA Category 1 credits. Fee: \$275. Contact: Cont. Med. Ed., Box 293 Mayo Memorial Bldg., 420 Delaware St., S.E., Minneapolis, MN 55455. Phone: (612) 373-8012.

November

Annual E. T. Bell Pathology Symposium: Problems in Gastrointestinal Pathology, Mayo Mem. Aud., U. of Minn., Minneapolis, Minn., Nov. 6. Contact: Cont. Med. Ed., Box 293 Mayo Memorial Bldg., 420 Delaware St., S.E., Minneapolis, MN 55455. Phone: (612) 373-8012.

Toxic and Poisoning Emergencies: Clinical Briefing, St. Paul-Ramsey Medical Ctr., St. Paul, Minn., Nov. 6. Fee: \$80.00. Contact: Cont. Med. Ed., St. Paul-Ramsey Med. Ctr., 640 Jackson St., St. Paul, MN 55101. Phone: (612) 221-3992.

Postgraduate Conference on Surgery: Pediatric Surgery, U. of Iowa Hosp. & Clinics, Nov. 6. AMA Category 1 credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

Frontiers in Medicine, First Annual Margaret G. Scheffer Memorial Lectureship, St. Joseph's Hospital, St. Paul, MN, Nov. 13. Fee: \$65. Contact: CME, St. Joseph's Hosp., 69 West Exchange St., St. Paul, MN 55102, (612) 291-3180.

VII Annual Childhood Cancer Workshop, U. of Iowa Hosp. & Clinics, Nov. 13. AMA Category 1 credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

Psychiatry Postgraduate Conference, U. of Iowa Hosp. & Clinics, Nov. 13, 14. AMA Category 1 credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

Advanced Trauma Life Support, U. of Iowa Hosp. & Clinics, Nov. 13-15. AMA Category 1 credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

Postgraduate Conference on Obstetrics and Gynecology, U. of Iowa Hosp. & Clinics, Nov. 17, 18. AMA Category 1 credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

Radiation Therapy Seminar, U. of Iowa Hosp. & Clinics, Nov. 19. AMA Category 1 credits. Contact: Richard M. Caplan,

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December

Cardiology Today, U. of Iowa Hosp. & Clinics, Dec. 1-4. AMA Category 1 credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

Ophthalmology Clinical Conference, U. of Iowa Hosp. & Clinics, Dec. 2. AMA Category 1 credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

The Evaluation and Current Treatment of Athletic Injuries: The Lower Extremity Kinetic Chain, Hyatt Regency O'Hare, Chicago, IL, Dec. 2-5. 17 hrs. AMA Category 1 credits. Contact: Kathy Johnson, CME, Box 48, MCV Station, Richmond, VA 23298.

Advanced Cardiac Life Support, U. of Iowa Hosp. & Clinics, Sioux City, IA, Dec. 4-6. AMA Category 1 credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

Behavioral Medicine and Primary Care in the '80's, Ilikai Hotel, Honolulu, HI, Dec. 4-11. 16 hrs. AMA Category 1 credits. Fee: \$300. Contact: Jeri McClain, Adm. Asst., USCSM, Off. for Academic Affairs, Columbia, SC 29208, (803) 777-7470.

Otolaryngology Clinical Conference, U. of Iowa Hosp. & Clinics, Dec. 11. AMA Category 1 credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, U. of Iowa College of Med., Iowa City, IA 52242.

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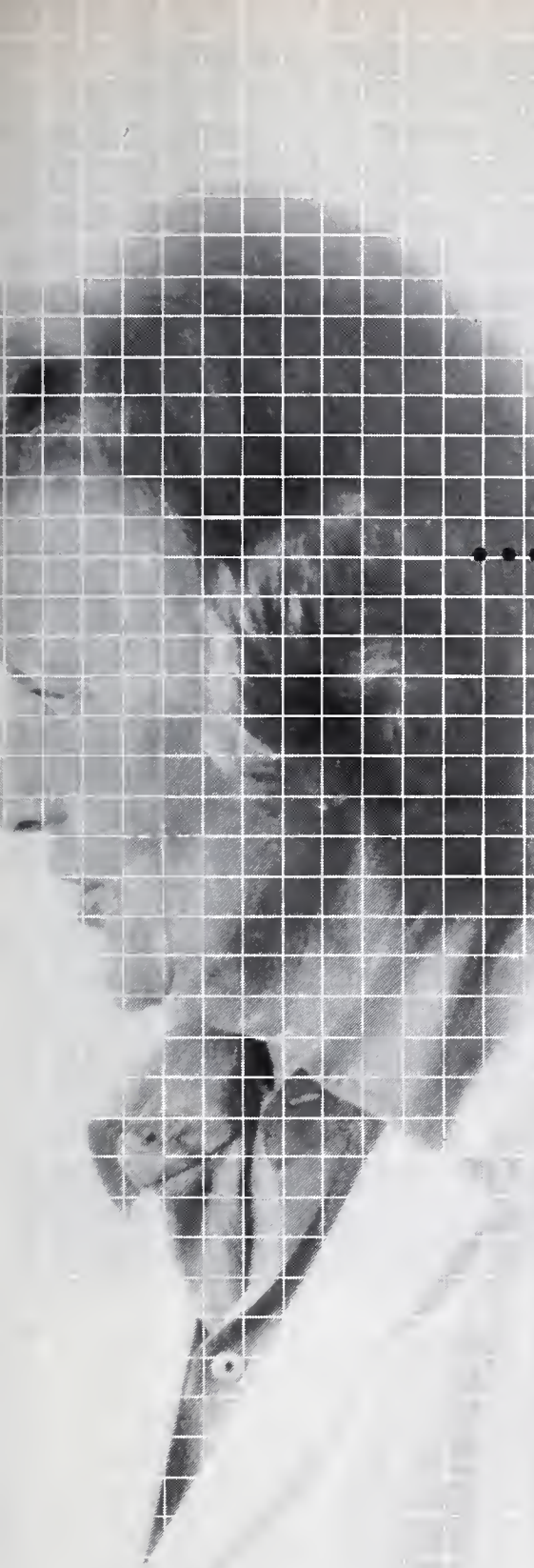
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Please see summary of product information on the following page.

VALIUM®(diazepam/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety. Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic. Symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis, stiff-man syndrome; convulsive disorders (not for sole therapy).

The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication, abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed, drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation. The clearance of Valium and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. **Adults:** Anxiety disorders, symptoms of anxiety, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed, adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. **Geriatric or debilitated patients:** 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) **Children:** 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

How Supplied: For oral administration, Valium scored tablets—2 mg, white; 5 mg, yellow; 10 mg, blue—bottles of 100* and 500; * Prescription Paks of 50, available in trays of 10 * Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25,† and in boxes containing 10 strips of 10.*

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EQUAGESIC—Abbreviated Summary

INDICATIONS: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective for the treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease or tension headache.

Final classification of the less-than-effective indications requires further investigation.

The effectiveness of Equagesic in long-term use, i.e. more than four months, has not been assessed by systematic clinical studies. The physician should periodically reassess usefulness of the drug for the individual patient.

CONTRAINDICATIONS: Equagesic should not be given to individuals with a history of sensitivity or severe intolerance to aspirin, meprobamate, or ethoheptazine citrate.

WARNINGS: Careful supervision of dose and amounts prescribed for patients is advised, especially with those patients with known propensity for taking excessive quantities of drugs. Excessive and prolonged use in susceptible persons, e.g., alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on or habituation to the drug. Where excessive dosage has continued for weeks or months, dosage should be reduced gradually rather than abruptly stopped, since withdrawal of a "crutch" may precipitate withdrawal reaction of greater proportions than that for which the drug was originally prescribed. Abrupt discontinuance of doses in excess of the recommended dose has resulted in some cases in the occurrence of epileptiform seizures.

Special care should be taken to warn patients taking meprobamate that tolerance to alcohol may be lowered with resultant slowing of reaction time and impairment of judgment and coordination.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with the use

of minor tranquilizers (meprobamate, chlorthalidone, and diazepam) during the first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant, they should communicate with their physicians about the desirability of discontinuing the drug. Meprobamate passes the placental barrier. It is present both in umbilical-cord blood and in near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breast-feeding patients, the drug's higher concentration in breast milk as compared to maternal plasma levels should be considered.

Preparations containing aspirin should be kept out of the reach of children. Equagesic is not recommended for patients 12 years of age and under.

PRECAUTIONS: Should drowsiness, ataxia, or visual disturbance occur, the dose should be reduced. If symptoms continue, patients should not operate a motor vehicle or any dangerous machinery.

Suicidal attempts with meprobamate have resulted in coma, shock, vasomotor and respiratory collapse, and anuria. Very few suicidal attempts were fatal, although some patients ingested very large amounts of the drug (20 to 40 gm). These doses are much greater than recommended. The drug should be given cautiously, and in small amounts, to patients who have suicidal tendencies. In cases where excessive doses have been taken, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Hyperventilation has been reported occasionally. Any drug remaining in the stomach should be removed and symptomatic treatment given. Should respiration become very shallow and slow CNS stimulants (e.g., caffeine, Metazolol or amphet-

mine, may be cautiously administered. If severe hypotension develops, pressor amines should be used parenterally to restore blood pressure to normal levels.

ADVERSE REACTIONS: A small percentage of patients may experience nausea with or without vomiting and epigastric distress. Dizziness occurs rarely when meprobamate and ethoheptazine citrate with aspirin is administered in recommended dosage. The meprobamate may cause drowsiness but, as a rule, this disappears as therapy is continued. Should drowsiness persist and be associated with ataxia, this symptom can usually be controlled by decreasing the dose, but occasionally it may be desirable to administer central stimulants such as amphetamine or mephentermine sulfate concomitantly to control drowsiness.

A clearly related side effect to the administration of meprobamate is the rare occurrence of allergic or idiosyncratic reactions. This response develops, as a rule, in patients who have had only 1-4 doses of meprobamate and have not had a previous contact with the drug. Previous history of allergy may or may not be related to the incidence of reactions.

Mild reactions are characterized by an itchy urticarial or erythematous, maculopapular rash which may be generalized or confined to the groin. Acute nonthrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema and fever have also been reported.

More severe cases, observed only very rarely, may also have other allergic responses, including fever, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case), and hyperthermia. Treatment should be symptomatic such as administration of epinephrine, antihistamine, and possibly hydrocortisone. Meprobamate should be stopped, and resumption of therapy should not be attempted.

Rare cases have been reported where patients receiving meprobamate suffered from aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia. In nearly every instance reported, other toxic agents known to have caused these conditions have been associated with meprobamate. A few cases of leukopenia during

continuous administration of meprobamate are reported, most of these returned to normal without discontinuation of the drug. Impairment of accommodation and visual acuity has been reported rarely.

OVERDOSE: Two instances of accidental or intentional significant overdosage with ethoheptazine citrate combined with aspirin have been reported. These were accompanied by symptoms of CNS depression, including drowsiness and light-headedness, with uneventful recovery. However, on the basis of pharmacological data, it may be anticipated that CNS stimulation could occur. Other anticipated symptoms would include nausea and vomiting. Appropriate therapy of signs and symptoms as they appear is the only recommendation possible at this time. Overdosage with ethoheptazine combined with aspirin would probably produce the usual symptoms and signs of salicylate intoxication. Observation and treatment should include induced vomiting or gastric lavage, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions.

DESCRIPTION: Each Equagesic tablet contains 150 mg meprobamate, 75 mg ethoheptazine citrate and 250 mg aspirin.

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*This drug has been evaluated as possibly effective for this indication.

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WYGESIC—Abbreviated Summary

INDICATION: For the relief of mild-to-moderate pain.

CONTRAINDICATION: Hypersensitivity to propoxyphene or to acetaminophen.

WARNINGS: CNS ADDITIVE EFFECTS AND OVERDOSE: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, or other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended. Toxic effects and fatalities have occurred following overdoses of propoxyphene alone or in combination with other CNS depressants. Most of these patients had histories of emotional disturbances or suicidal ideation or attempts, as well as misuse of tranquilizers, alcohol, or other CNS-active drugs. Caution should be exercised in prescribing large amounts of propoxyphene for such patients (see **Management of Overdosage**).

DRUG DEPENDENCE: Propoxyphene can produce drug dependence characterized by psychic dependence and less frequently physical dependence and tolerance. It will only partially suppress the withdrawal syndrome in individuals physically dependent on morphine or other narcotics. The abuse liability of propoxyphene is qualitatively similar to codeine's although quantitatively less, and propoxyphene should be prescribed with the same degree of caution appropriate to the use of codeine.

USAGE IN AMBULATORY PATIENTS: Propoxyphene may impair the mental and/or physical abilities required for potentially hazardous tasks, e.g. driving a car or operating machinery. Patients should be cautioned accordingly.

USAGE IN PREGNANCY: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. **INSTANCES OF WITHDRAWAL SYMPTOMS IN THE NEONATE HAVE BEEN REPORTED FOLLOWING USAGE DURING PREGNANCY.** Therefore, propoxyphene should not be used in pregnant women unless, in the

judgement of the physician, the potential benefits outweigh the possible hazards.

USAGE IN CHILDREN: Propoxyphene is not recommended for children because documented clinical experience has been insufficient to establish safety and a suitable dosage regimen in the pediatric group.

PRECAUTIONS: Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine. The CNS depressant effect of propoxyphene may be additive with other CNS depressants, including alcohol.

ADVERSE REACTIONS: The most frequent adverse reactions are dizziness, sedation, nausea, and vomiting. These seem more prominent in ambulatory actions may be alleviated if the patient lies down.

Other adverse reactions include constipation, abdominal pain, skin rashes, light-headedness, headache, weakness, euphoria, dysphoria, and minor visual disturbances. The chronic ingestion of propoxyphene in doses over 800 mg per day has caused toxic psychoses and convulsions. Cases of liver dysfunction have been reported.

DRUG INTERACTIONS: Propoxyphene in combination with alcohol, tranquilizers, sedative-hypnotics, and other CNS depressants has an additive depressant effect. Patients taking this drug should be advised of the additive effect and warned not to exceed the dosage recommended (see **Warnings**). Confusion, anxiety, and tremors have been reported in a few patients receiving propoxyphene concomitantly with orphenadrine.

MANAGEMENT OF OVERDOSAGE: SYMPTOMS: The manifestations of serious overdosage with propoxyphene are similar to those of narcotic overdosage and include respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, cyanosis), extreme somnolence progressing to stupor or coma, pupillary constriction, and circulatory collapse. In addition to these characteristics, which are reversed by narcotic antago-

nists such as naloxone, there may be other effects. Overdoses of propoxyphene can cause delay of cardiac conduction as well as focal or generalized convulsions, a prominent feature in most cases of severe poisoning. Cardiac arrhythmias and pulmonary edema have occasionally been reported and apnea, cardiac arrest, and death have occurred.

Symptoms of massive overdosage with acetaminophen may include nausea, vomiting, anorexia, and abdominal pain, beginning shortly after ingestion and lasting for 12 to 24 hours. However, early recognition may be difficult since early symptoms may be mild and nonspecific. Evidence of liver damage is usually delayed. After the initial symptoms, the patient may feel less ill; however, laboratory determinations are likely to show a rapid rise in liver enzymes and bilirubin. In case of serious hepatotoxicity, jaundice, coagulation defects, hypoglycemia, encephalopathy, coma, and death may follow. Renal failure due to tubular necrosis, and myocardialopathy, have also been reported.

Ingestion of 10 grams or more of acetaminophen may produce hepatotoxicity. A 13-gram dose has reportedly been fatal.

TREATMENT: Primary attention should be given to the reestablishment of adequate respiratory exchange through provision of a patent airway and institution of assisted or controlled ventilation. The narcotic antagonists, naloxone, nalorphine, and levallorphan, are specific antidotes against the respiratory depression produced by propoxyphene. An appropriate dose of one of these antagonists should be administered preferably, I.V., simultaneously with efforts at respiratory resuscitation and the antagonist should be repeated as necessary until the patient's condition remains satisfactory. In addition to a narcotic antagonist, the patient may require careful titration with an anticonvulsant to control seizures. Analeptic drugs (e.g. caffeine or amphetamine) should not be used because of their tendency to precipitate convulsions.

Oxygen, IV fluids, vasopressors and other supportive measures should be used as indicated. Gastric lavage may be helpful. Activated charcoal can absorb a significant amount of ingested propoxyphene. Dialysis is of little value in poisoning by propoxyphene alone. Acetaminophen is rapidly absorbed and efforts to remove the drug from the body should not be delayed. Copious gastric lavage and/or induction of emesis may be indicated. Activated charcoal is probably ineffective unless administered almost immediately after acetaminophen ingestion. Neither forced diuresis nor hemodialysis appears to be effective in removing acetaminophen. Since acetaminophen in overdose may have an antidiuretic effect and may produce renal damage, administration of fluids should be carefully monitored to avoid overload. It has been reported that mercaptamine (cysteamine) or other thiol compounds may protect against liver damage if given soon after overdosage (8-10 hours). N-acetylcysteine is under investigation as a less toxic alternative to mercaptamine, which may cause anorexia, nausea, vomiting, and drowsiness. Appropriate literature should be consulted for further information (JAMA 237:2406-2407, 1977).

Clinical and laboratory evidence of hepatotoxicity may be delayed up to one week. Acetaminophen plasma levels and half-life may be useful in assessing the likelihood of hepatotoxicity. Serial hepatic enzyme determinations are also recommended.

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Forty-Nine Year Old Caucasian Male With Right Upper Quadrant Pain

Jean A. Eller, M.D.*
Discusser

John F. Barlow, M.D.**
Editor

Case No. 859 957

This 49-year-old Caucasian male house painter was referred to a specialist in internal medicine because of an increased serum iron.

Two years prior to admission, the patient had had two episodes of sharp right upper quadrant pain which did not recur until four months prior to admission when the patient began to have similar right upper quadrant pain which was unrelated to meals, type of food, or position and did not radiate into the back, shoulders or groin. Occasionally the pain was sharp and nagging but was always present. The patient had had a gallbladder workup one year prior to admission with no abnormal findings. There was no history of excess alcohol intake, iron intake, transfusions or family history of diabetes, skin pigmentation or heart disease. The patient had had a rash after exposure to penicillin in the past. A review of systems was negative except for a 10 to 15 pound weight loss over the past 6 months and a history of chronic seminal vesiculitis, not requiring current therapy.

PHYSICAL EXAMINATION: Temperature 98°F, pulse 80/minute and regular; respirations 16/minute and regular; blood pressure 142 systolic and 88 diastolic. The patient was in no acute distress. There was no significant skin pigmentation. Examination of the head and neck was unremarkable. The lungs were clear to auscultation and percussion. The heart was not enlarged and had a regular rhythm with no heart murmurs or abnormal sounds. There was some tenderness over the right upper quadrant and the liver was felt to be 2 to 3 cm. below the costal margin. The spleen was not palpable. The extremities and neurologic examination were within normal limits.

LABORATORY DATA: Urinalysis—dark to straw colored, clear; specific gravity 1.022; pH 5.0, negative for protein, glucose, ketone bodies, bile and hemoglobin; sediment 0-1 white cells/hpf; few calcium oxalate crystals. Hgb. 16.9 gm/dl; hct. 40 vol/dl with normal red cells indices. Total leukocyte count 6,000/mm³ (6.0

x 10⁹/L) with 53% segmented neutrophils, 2% neutrophilic bands, 45% lymphocytes. The red cells were normochromic, normocytic and the platelets were normal in number and morphology on the smear. A platelet count was 173,000/mm³ (173 x 10⁹/L). Prothrombin time and partial thromboplastin time were within normal limits. Lactic dehydrogenase (LDH), alkaline phosphatase, aspartate aminotransferase (SGOT), total bilirubin, total protein, calcium, phosphorus, glucose, blood urea nitrogen, creatinine, uric acid, cholesterol, and T₄ by radioimmunoassay were within normal limits. Blood pH and electrolytes were within normal limits. A serum electrophoresis showed no diagnostic abnormality. A serum iron was 175 ugs/dl (normal 50-180 ug/dl), transferrin 230 ug/dl (normal 200-400 ug/dl), percent saturation of transferrin 76% (normal 20-40%), blood lead 12 ug/dl (normal 0-40 ug/dl), serum ferritin 2,000 ug/nl (normal 20-200 ng/nl). A desferrioxamine test was elevated at 5.7 mg/24 hr. (normal 0.15 mg/24 hr.). An electrocardiogram showed marked sinus bradycardia. A radioisotope liver and spleen scan showed mild splenomegaly. A cholecystogram showed cholelithiasis and a chest film was negative. A diagnostic procedure was performed.

DR. ELLER: The salient features in this case are right upper quadrant pain, fifteen pound weight loss, mild hepatosplenomegaly and elevated serum iron studies in a previously healthy 40-year-old man.

The differential diagnosis might begin with plumbism since this man has been a painter. This disease follows a long term cumulative exposure with lead deposition in the bones. Symptoms are colic, wandering abdominal pains with spasm and rigidity. These episodes are aggravated by alcohol and infection. Patients may also have peripheral neuropathies. Anemia is the rule with basophilic stippling. Our patient has polycythemia as well as localized abdominal pain. A normal serum lead helps exclude plumbism.

Alcoholism is often accompanied by iron over-

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load. These patients present with the usual stigmata of cirrhosis plus secondary elevation of serum iron studies. Our patient gives no history of heavy alcohol intake, so we can probably exclude this as a cause of his illness. His normal liver function tests, bleeding studies, and red cell indices help support this assumption.

Cholelithiasis certainly can cause right upper quadrant pain, but the pain described above is not classic for gallbladder disease, i.e. there is no association with food intake. Neither could gallstones alone cause elevated iron studies. Cholelithiasis could be an incidental finding.

Thalassemias may cause elevated iron studies with organomegaly. Anemia and abnormal hemoglobin electrophoresis should be present. Since it is an hereditary disease, there should be a history in the family as well. We will not consider thalassemia since our patient had a normal hemoglobin electrophoresis and no anemia.

It might be interesting to know whether the patient spent a significant portion of his life taking dietary supplementation with iron. This is practiced in some countries, and may aggravate incipient iron overload.

Since our patient has some markedly elevated iron studies, a discussion of the metabolism of this heavy metal is appropriate. After oral ingestion, iron is stabilized in the stomach by hydrochloric acid and bound to chelators. This creates soluble ferrous (Fe^{+2}) iron which is transported to the small intestine. Absorption is maximal in the upper duodenum and decreases during passage through the upper gastrointestinal tract. Ascorbic acid, alcohol and sugars facilitate iron absorption. Several physiologic conditions such as anemia, hypoxia, decreased iron stores and increased erythropoiesis also augment iron absorption, but the exact mechanisms are unknown. Other heavy metals such as manganese and cobalt impair iron absorption. After arriving in the intestine, iron is removed from the lumen by the mucosal cell, bound to intracellular proteins and then released to the blood on transferrin by which iron is carried to body cells and becomes storage iron or functioning iron in hemoglobin or myoglobin.

Transferrin is a polypeptide which tightly binds iron by means of an anion, probably HCO_3^{-2} . This transport form exchanges iron with all body cells and goes directly into the bone marrow to release its iron for erythropoiesis. It can deliver iron to parenchymal tissues but generally not to the reticuloendothelial (RE) system. Transferrin is elevated in pregnancy and estrogen therapy, decreased in inflammation and iron overload. This transport molecule is normally one-third saturated with iron.

Hemosiderin is a storage form of iron, usually located in the RE system. It can occasionally be deposited in the bone marrow, liver and lung. Hemosiderosis can occur with excessive intake of oral iron.

Ferritin is an iron-protein complex made in the liver and RE system. It is the major iron storage protein and is relatively unsaturated with iron. The serum ferritin concentration varies directly with body iron stores so it is an excellent indicator of total body stores. Ferritin is actually a conglomeration of isoferritins, which refer to tissue specific forms of ferritin.³

The major regulator of iron is the body requirement². Several theories attempt to explain the mechanism for this. The presence of ferritin in the mucosal cell may prevent further iron uptake. Iron-loaded mucosal cells may slough into the bowel if excess iron is present. It is well documented that in iron deficiency, iron absorption increases greatly. Hormonal feedback via the serum iron-protein complexes may regulate intestinal iron absorption as well.¹

Body iron is distributed as follows: hemoglobin (70%), myoglobin (10%), and total storage forms (30%). Approximately one milligram is absorbed daily. There is no major excretory pathway for iron. Iron is essentially locked in the body once absorbed. The average man in the United States has five grams of body iron, while women average two or three grams.

True iron overload originates from one or more of three mechanisms.

1. increased mucosal absorption (e.g. idiopathic hemochromatosis)
2. increased dietary intake (e.g. medicinal, food fortification)
3. excessive parenteral treatments (e.g. transfusions, iron injections)

Primary idiopathic hemochromatosis (IHC) is defined as an increase in total body iron with iron deposition in parenchymal tissues causing damage.⁴ The classic triad, first described in 1935 by Sheldon, consists of cirrhosis of the liver, diabetes mellitus and hyperpigmentation of the skin. The liver, pancreas, heart, pituitary, gonads, and thyroid can have iron deposition. IHC is an inborn error of metabolism transmitted as an autosomal recessive trait. Prevalence is about 1 per 10,000 persons. Males are affected ten times more commonly than women. This is due to the fact that women have considerable iron losses during pregnancy, lactation, and menses; and, therefore, if the disease surfaces, it does so at an older age. Patients may present with diabetes mellitus, congestive heart failure, arrhythmias or polyarthropathy.

Skin hyperpigmentation in the IHC is described as a metallic grey hue and is secondary to increased melanin deposition in the basal layer of the epidermis. Diabetes can be refractory to treatment in this syndrome, and is probably a result of iron in the pancreas as well as glucose intolerance secondary to hepatic dysfunction. The glucagon function of the pancreas remains intact.

Cardiac complications can be serious. Congestive failure and rapid atrial arrhythmias are most common.⁵ These abnormalities are treated conventionally and improve greatly with treatment of the hemochromatosis.

The part of the endocrine system involved most often is the anterior pituitary gland resulting in hypogonadism, and less often thyroid and adrenal insufficiency. Testicular atrophy, impotence, and menstrual irregularities may occur.

Arthropathy is seen in older patients and unfortunately does not improve with treatment. Pseudogout, chondrocalcinosis and degenerative arthritis all play a part in the arthropathy, which tends to affect the metacarpal-phalangeal and proximal interphalangeal joints first. Weight bearing joints are affected later in the disease.

The liver is the first organ damaged. Periportal fibrosis and later nodular cirrhosis characterize the histologic picture along with massive iron deposition. Liver function tests are often normal and there are no fatty changes as in alcoholic cirrhosis with iron overload. Hepatoma occurs frequently in advanced stages and may be seen as long as sixteen years after hemochromatosis has been detected. The incidence does not decrease with treatment of the iron overload.

It is important to distinguish between IHC and alcoholic cirrhosis because treatment is necessary in the former only. Patients with alcoholic cirrhosis have iron overload because alcohol is known to increase iron absorption. Wines contain a large amount of iron and destruction of hepatic cells leads to increased uptake of iron by Kupffer cells.

The mechanism of cellular damage of iron is thought to be secondary to the formation of free radicals as iron goes from the ferric to ferrous form within the cell.³ Free radicals oxidize tissues and rupture membranes of lysosomes with release of enzymes leading to inflammation and fibrosis. Iron may directly stimulate collagen formation as well.

Some researchers feel IHC may be a multisystem disease of abnormal iron uptake.⁷ For example, in fully treated patients who have cirrhosis and elevated iron studies, liver iron uptake is significantly higher than controls. Curiously, in IHC the bone marrow has conspicuously little iron while parenchymal cells are heavily laden with it. In secondary hemochro-

matosis from multiple blood transfusions or excessive iron intake, the RE system and bone marrow are appropriately saturated with iron. A defect in ferritin synthesis may explain the pathogenesis of IHC.

Of primary concern is the inheritance pattern of IHC since early detection and treatment of homozygous persons may prevent organ damage. Chromosome number six has two alleles associated with the HLA grouping which are responsible for the inheritance of IHC. Heterozygous patients have only biochemical changes and need not be treated unless aggravating factors such as alcoholism are operating. Efforts have been made to correlate serum iron studies with the zygosity of patients so that the homozygous person could be found and spared the expense and technical difficulty of being expensively HLA typed.^{8,9,10} Serum iron has a 25% false negative rate in detecting IHC patients, the percent saturation of transferrin has a 33% false positive rate, while serum ferritin has only a 1.8% false positive rate and good sensitivity in homozygotes. Nevertheless serum iron, percent saturation of transferrin and serum ferritin are all recommended as screening tests for all first and second degree relatives.⁸ The serum ferritin appears to be the most accurate of the three tests.

HLA testing is only of value as a prognosticator since therapy and definitive diagnosis of hemochromatosis depends on estimating total body iron stores. The following methods reflect these iron stores: serum iron, percent saturation of transferrin, serum ferritin, desferroxamine chelation test, amount of stainable hepatic iron, chemical estimation of iron and quantitative phlebotomy.

Treatment of IHC consists of weekly 500 cc. phlebotomy until the hemoglobin approaches 11.0 gm/dl. Each unit of blood removes about 250 mg of iron. 80-100 phlebotomies may be required before a good therapeutic effect is achieved. Maintenance therapy required four to six phlebotomies per year. Chelation therapy with desferroxamine removes too little iron to be of value in IHC. Retrospective studies show a marked improvement in five year survival in those patients who receive treatment compared to those untreated.

In summary, our patient has markedly elevated iron studies and no history of alcoholism or hereditary anemia.

Dr. Jean Eller's Diagnosis Idiopathic Hemochromatosis

DR. BARLOW: The diagnostic procedure performed was a liver biopsy. The first slide demonstrates that the liver architecture is altered from normal to a nodular pattern by bands of fibrous

tissue separating regenerative nodules. (Fig. 1) In addition to this pattern consistent with early cirrhosis, there is a prominent brown granular refractile pigment in the parenchymal cells as well as in the bile duct epithelium in the portal spaces. On iron stain (Prussian blue reaction) the pigment gives the characteristic reaction for iron and proves the pigment is hemosiderin. This high power hematoxylin and eosin stain shows the typical hemosiderin granules in the liver cells. (Fig. 2) A quantitative iron was also taken in this case and was markedly elevated. I could not locate the exact report.

FINAL ANATOMIC DIAGNOSIS HEMOCHROMATOSIS WITH EARLY CIRRHOSIS

*DR. BARKER: I do not have the quantitative iron report with me but it was markedly elevated in the range of hemochromatosis.

DR. BARLOW: I would like to mention that it is important to diagnose this disease early, not only to prevent tissue damage in the patient in whom it is diagnosed, but also in the patient's relatives and children.

**DR. LOREN TSCHETTER: I agree. Blood relatives can be detected often by using serum iron, serum transferrin, and serum ferritin. However, these are not always markedly elevated and I have seen a recent case in which a relative of a patient with hemochromatosis had a normal serum iron and ferritin but had appreciable iron noted on a liver biopsy obtained at laparotomy performed for another disease. The best way to demonstrate inheritance of hemochromatosis is by doing histocompatibility testing (HLA typing). However, this is very expensive.

DR. BARKER: I would like to point out that many cases of hemochromatosis are not diagnosed by the classical triad of pigmentation of the skin, cirrhosis of the liver, and diabetes mellitus or by the clinical picture of a "bronze diabetes". They are often detected much earlier than this. I have seen several patients diagnosed by elevated serum iron levels on a chemistry panel or other associated symptoms such as arthritis.

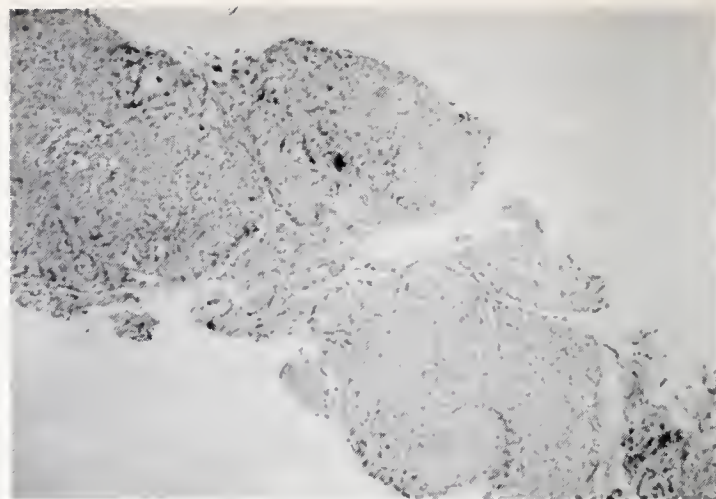


Figure 1

Note early cirrhosis with fibrous bands and regenerative nodules 100x.

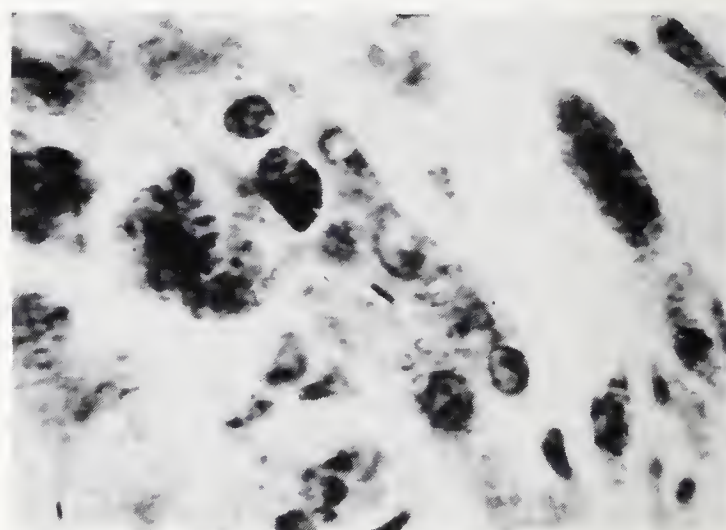


Figure 2

Abundant hemosiderin pigment in macrophages and hepatic parenchymal cells.

***DR. K. H. WEGNER: Does anyone know about the continuous chelation therapy with desferroxime employed in certain patients who carry a small instrument with them at all times?

DR. TSCHETTER: This is often utilized in patients with thalassemia major who suffer iron overload but cannot tolerate phlebotomy, a more preferable therapy for hemochromatosis.

****DR. W. PUTNAM: How does one separate porphyria cutanea tarda with iron overload from hemochromatosis.⁷

DR. BARKER: The characteristic photosensitive skin rash of porphyria cutanea tarda may be subtle but these patients usually excrete large quantities of uroporphyrin I.

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**** Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital, Sioux Falls, SD.

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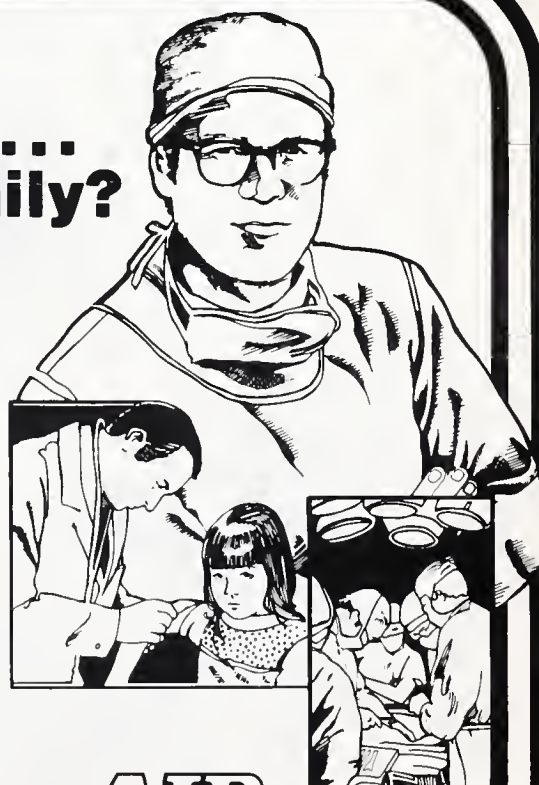
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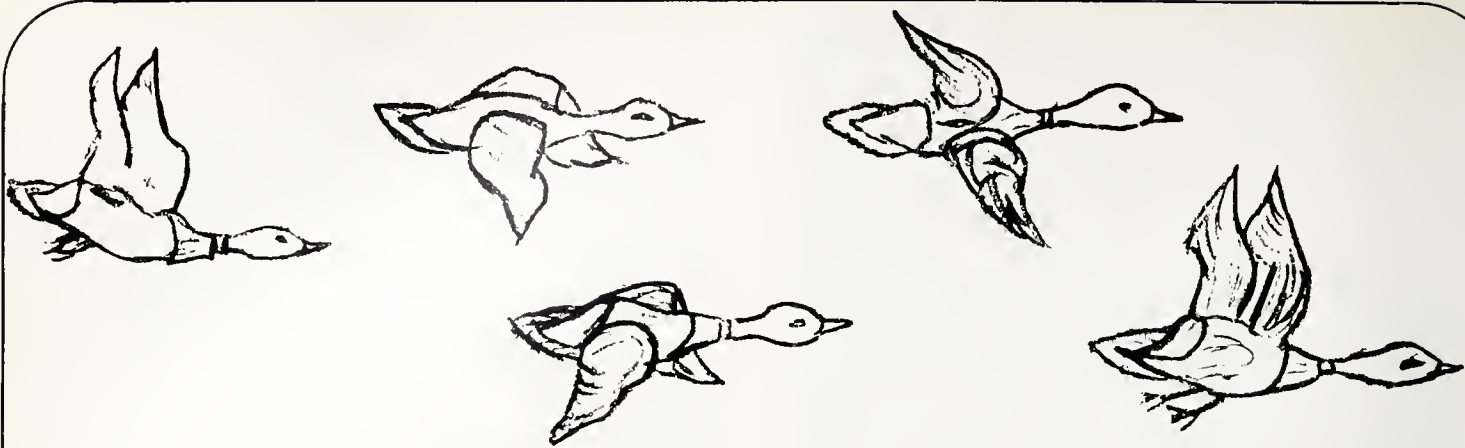
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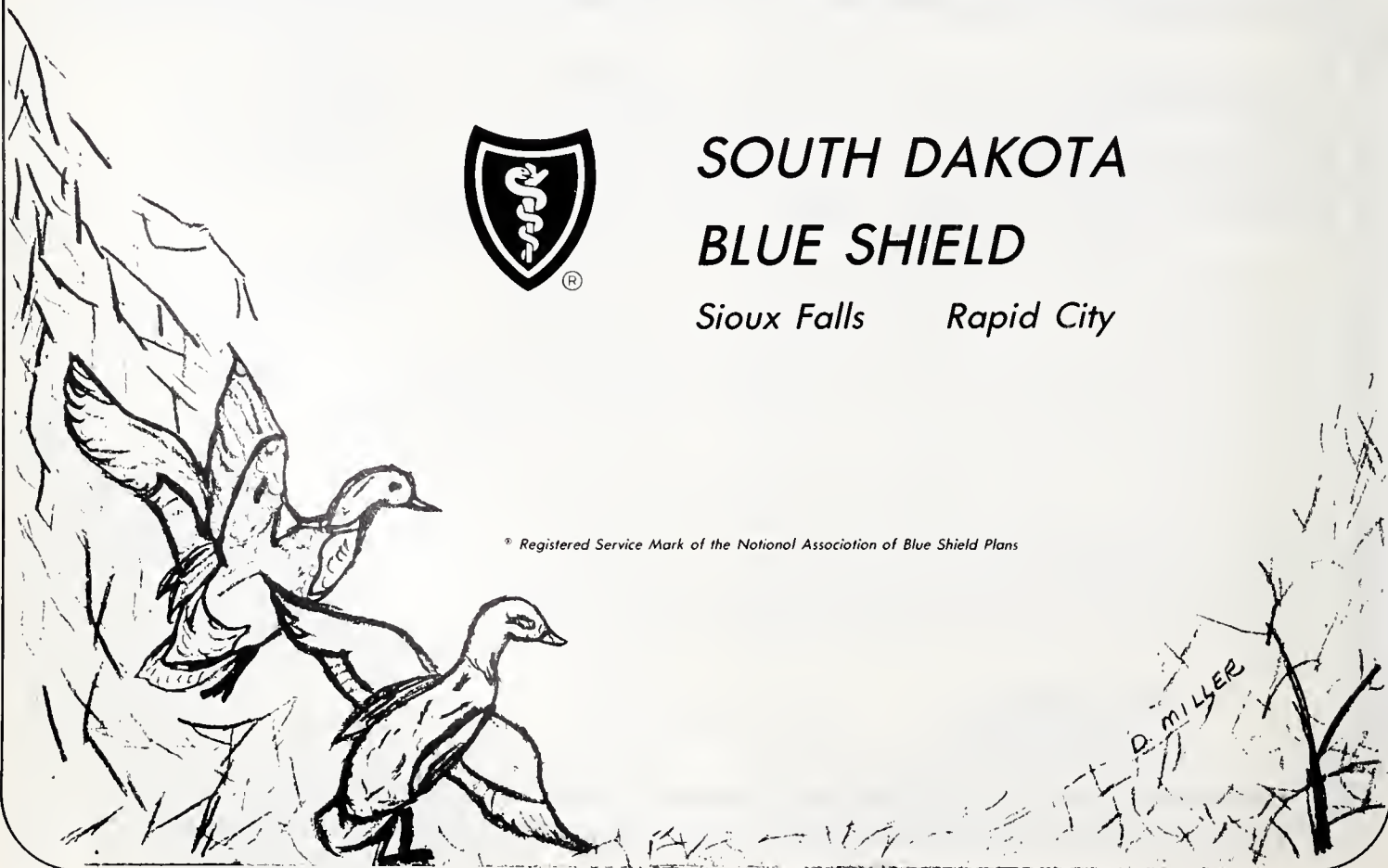
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SDAFP Receives Membership Award

For the third consecutive year, our Academy received a national award for being one of the states to have 100% of potential family practice resident members enrolled.

This award, a plaque given by the AAFP Membership Commission, was received by President Gene Nemer at the recent national meeting in Las Vegas. The plaque hangs in the conference room of the new Family Practice Center at 2300 S. Dakota Ave., Sioux Falls.

SDAFP Members "Designated to Provide Family Practice Planning Input" for CME Programs Requesting Prior Approval:

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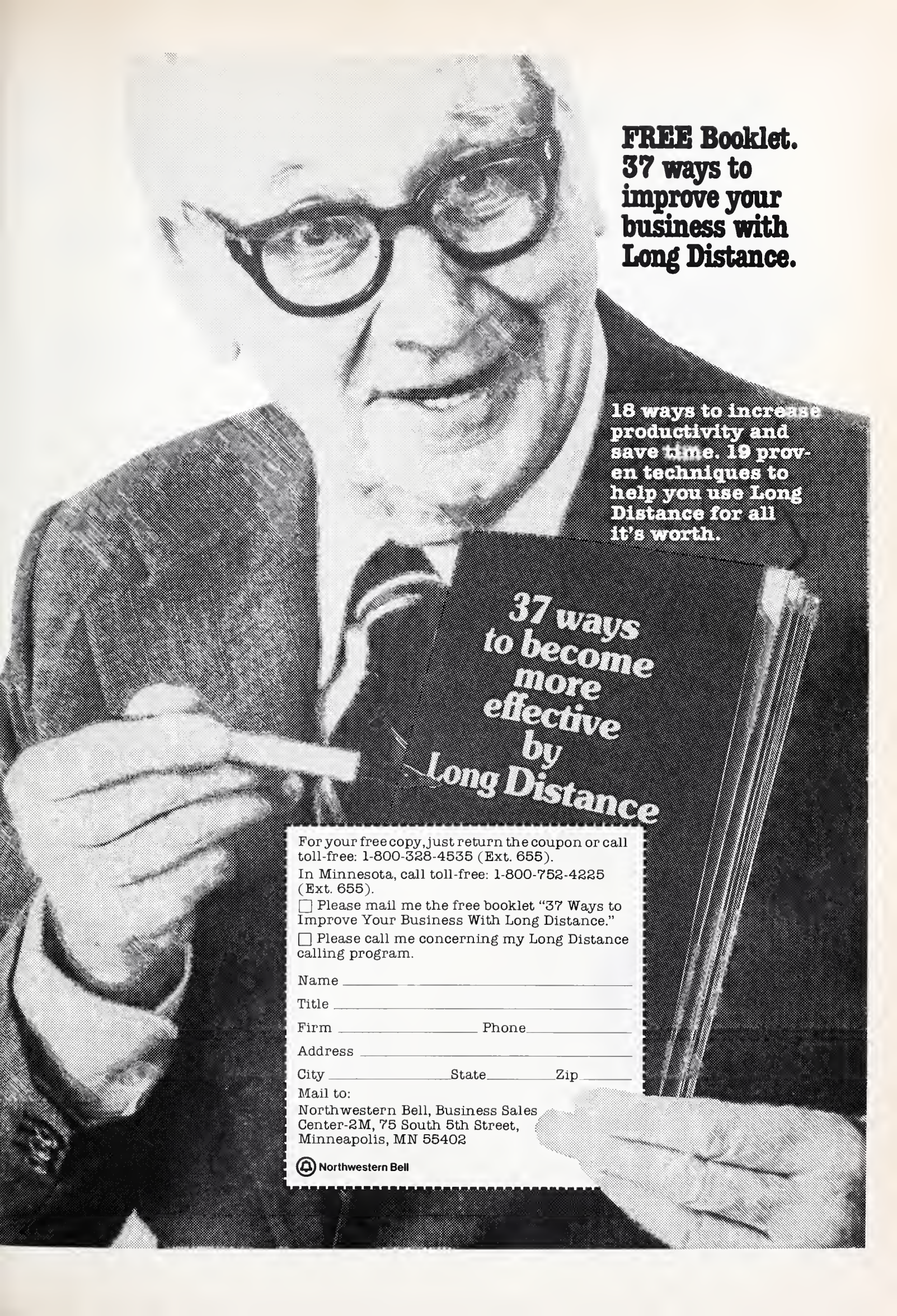
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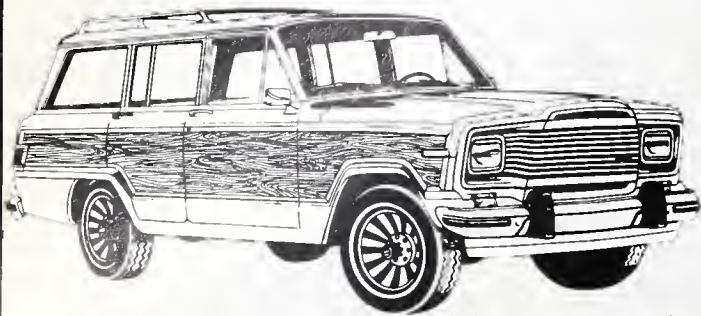
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A Critical Time For The University Of South Dakota School Of Medicine

The University of South Dakota School of Medicine has now graduated five "MD" degree classes (1977-1981). Of the 210 graduates, most have chosen primary care residencies and many have chosen Family Practice residencies—appropriate for a school with a legislative mandate to be family practice oriented.

It is too soon to evaluate the schools impact on physician needs in South Dakota. However, most people feel that the 4 year medical school has been a major reason for the significant increase in physicians practicing in South Dakota since 1970. There were 502 licensed and practicing MD's in South Dakota in 1970, 558 in 1975 and 815 in 1981. The University of South Dakota School of Medicine is graduating well-trained medical students destined to be excellent physicians. This is a record that the School of Medicine has had since its very beginning. The school needs our strong support. I think it is time for each of us to let our support be known! Why? Higher education budgets in South Dakota are suffering from inflation just like all segments of our society. The medical school is no exception. The school has never been funded for the original staffing levels projected in 1973 when planning for the four-year school was initiated.

I realize that we cannot change the decisions already made in terms of budget allocations for the school. However, it is critical that the people of South Dakota, including the Board of Regents, our governor, and our legislators, know that we feel our medical school is important and vital to health care delivery in South Dakota now and in the future. Otherwise, other budget items may take priority when it is time for dollars to be allocated by the state legislature. So, at every opportunity, give your support to "your school", a quality medical school.



Specific needs in the next year are additional dollars (\$250,000) to replace federal capitation money which had an early termination in July of this year. Dean Hollerman has requested funding for twenty-three faculty, to ensure our continued accreditation and to bring our staffing up to the original projected needs of the school. Ten faculty are needed in basic science and 13 in clinical departments. Additional funds are also needed in all areas because of increases in operational costs.

It is also possible that if additional funding dollars are not available the school's accreditation may be jeopardy and the multi-campus concept (Vermillion, Yankton, Sioux Falls, and Rapid City) will be unable to be fully realized or maintained.

In 1979, when the school was given full accreditation for a two year period emphasis was made by the accreditation team concerning the continuation of adequate budget dollars and sufficient faculty. The 1981 LCME accreditation team will make a site visit to the school this month.

This is a critical time for medical education in South Dakota, in my opinion. The faculty is willing to do all it can to continue educating doctors for South Dakota. We, as South Dakota State Medical Association Members, need to continue our support and let it be known. It takes only a few careless comments to create the attitude that we don't care. The fact is that we must care enough to give the very best education to the future physicians of South Dakota.

Sincerely yours,

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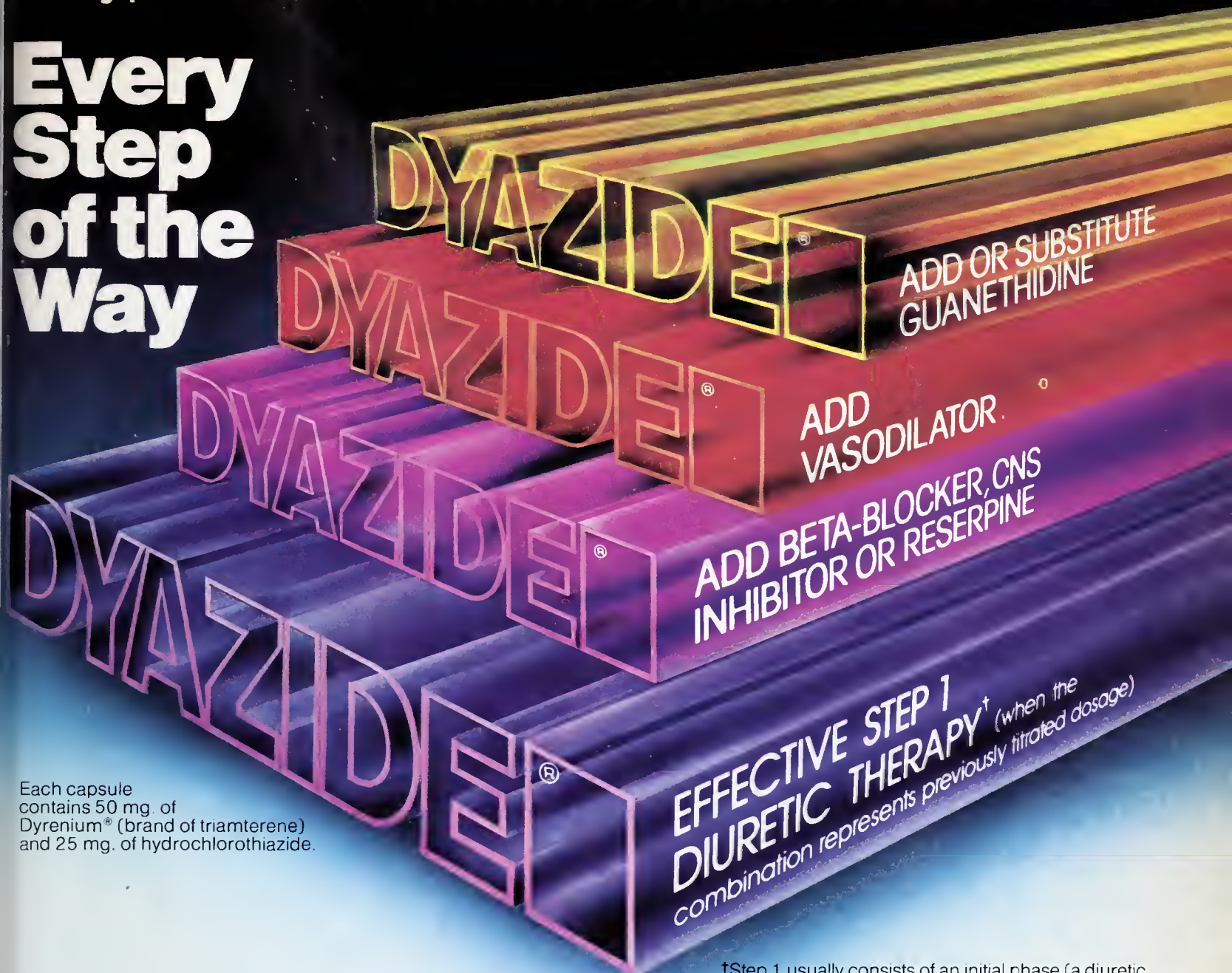
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triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K⁺ frequently, both can cause K⁺ retention and elevated serum K⁺. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other, serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased

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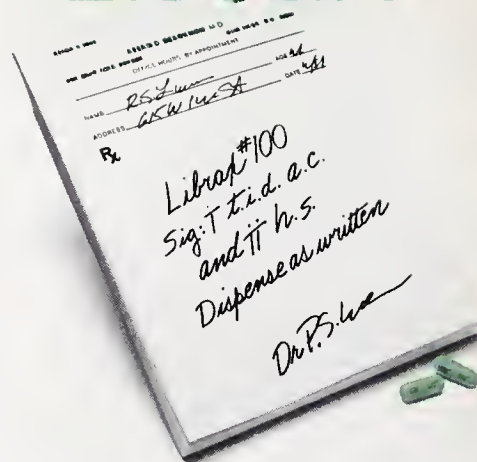
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Contraindications: Glaucoma, prostatic hypertrophy, benign bladder neck obstruction, hypersensitivity to chlordiazepoxide HCl and/or clidinium bromide

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage, withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.

As with all anticholinergics, inhibition of lactation may occur

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially, increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression, suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated, avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction, changes in EEG patterns may appear during and after treatment, blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, i.e., dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.



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Irritable BOWEL SYNDROME*

Artist's concept of myoelectrical slow waves of the colon which seem to determine the frequency of colonic motor activity.

A visible difference in myoelectric rhythms of the colon

Studies reveal an increased frequency of 3-cycles-per-minute slow wave basic electrical activity in the colons of patients with IBS—a significant difference in basic colonic rhythm patterns from normal subjects.^{1,2} These findings suggest a physiological basis for the spasm and hypermotility characteristic of IBS. The role of severe anxiety in triggering or aggravating such symptoms has long been recognized. Consequently, treatment should focus on both aspects of the problem.

Librax: A logical choice for patients with IBS

Logical, because the antimotility-antispasmodic actions of the Quarzan® (clidinium bromide/Roche) component of Librax can help to relieve the distressing abdominal symptoms associated with IBS.* Logical, because the antianxiety actions of the Librium® (chlordiazepoxide HCl/Roche) component can help to reduce the excessive anxiety that can contribute to IBS flare-ups.

References: 1. Sullivan MA, Cohen S, Snape WJ: *N Engl J Med* 298:878-883, Apr 20, 1978
2. Snape WJ et al: *Gastroenterology* 72: 383-387, Mar 1977.

Specify **Librax**®

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium Br.

Antianxiety/Antisecretory/Antispasmodic

*Librax has been evaluated as possibly effective for this indication. Please see summary of prescribing information on facing page.

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(quinine sulfate tablets)

each tablet contains quinine sulfate 260 mg



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Nocturnal recumbency leg muscle cramping is frequently an unwelcome bedfellow for many patients—especially those with arthritis, diabetes, or peripheral vascular disease... consider Quinamm... simple, convenient dosage—usually just one tablet at bedtime... can provide restful, welcome sleep without night leg cramps.

Quinamm™

(quinine sulfate tablets)

CAUTION Federal law prohibits dispensing without prescription
BRIEF SUMMARY

INDICATIONS AND USAGE

For the prevention and treatment of nocturnal recumbency leg muscle cramps

CONTRAINDICATIONS

Quinamm may cause fetal harm when administered to a pregnant woman. Congenital malformations in the human have been reported with the use of quinine, primarily with large doses (up to 30 g) for attempted abortion. In about half of these reports the malformation was deafness related to auditory nerve hypoplasia. Among the other abnormalities reported were limb anomalies, visceral defects, and visual changes. In animal tests, teratogenic effects were found in rabbits and guinea pigs and were absent in mice, rats, dogs, and monkeys. Quinamm is contraindicated in women who are or may become pregnant. If this drug is used during pregnancy, or if the patient becomes pregnant while taking this drug, the patient should be apprised of the potential hazard to the fetus. Because of the quinine content, Quinamm is contraindicated in patients with known quinine hypersensitivity and in patients with glucose-6-phosphate dehydrogenase (G-6-PD) deficiency.

Since thrombocytopenic purpura may follow the administration of quinine in highly sensitive patients, a history of this occurrence associated with previous quinine ingestion contraindicates its further use. Recovery usually occurs following withdrawal of the medication and appropriate therapy.

This drug should not be used in patients with tinnitus or optic neuritis or in patients with a history of blackwater fever.

WARNINGS

Repeated doses or overdosage of quinine in some individuals may precipitate a cluster of symptoms referred to as cinchonism. Such symptoms, in the mildest form, include ringing in the ears, headache, nausea, and slightly disturbed vision, however, when medication is continued or after large single doses, symptoms also involve the gastrointestinal tract, the nervous and cardiovascular systems, and the skin.

Hemolysis (with the potential for hemolytic anemia) has been associated with a G-6-PD deficiency in patients taking quinine. Quinamm should be stopped immediately if evidence of hemolysis appears.

If symptoms occur, drug should be discontinued and supportive measures instituted. In case of overdosage, see OVERDOSAGE section of prescribing information.

PRECAUTIONS

General

Quinamm should be discontinued if there is any evidence of hypersensitivity (See CONTRAINDICATIONS). Cutaneous flushing, pruritus, skin rashes, fever, gastric distress, dyspnea, ringing in the ears, and visual impairment are the usual expressions of hypersensitivity, particularly if only small doses of quinine

have been taken. Extreme flushing of the skin accompanied by intense, generalized pruritus is the most common form. Hemoglobinuria and asthma from quinine are rare types of idiosyncrasy.

In patients with atrial fibrillation, the administration of quinine requires the same precautions as those for quinidine. (See Drug Interactions.)

Drug Interactions

Increased plasma levels of digoxin and digitoxin have been demonstrated in individuals after concomitant quinine administration. Because of possible similar effects from use of quinine, it is recommended that plasma levels for digoxin and digitoxin be determined for those individuals taking these drugs and Quinamm concomitantly.

Concurrent use of aluminum-containing antacids may delay or decrease absorption of quinine.

Cinchona alkaloids, including quinine, have the potential to depress the hepatic enzyme system that synthesizes the vitamin K-dependent factors. The resulting hypoprothrombinemic effect may enhance the action of warfarin and other oral anticoagulants.

The effects of neuromuscular blocking agents (particularly pancuronium, succinylcholine, and tubocurarine) may be potentiated with quinine and result in respiratory difficulties.

Urinary alkalinizers (such as acetazolamide and sodium bicarbonate) may increase quinine blood levels with potential for toxicity.

Drug Laboratory Interactions

Quinine may produce an elevated value for urinary 17-ketogenic steroids when the Zimmerman method is used.

Carcinogenesis, Mutagenesis, Impairment of Fertility

A study of quinine sulfate administered in drinking water (0.1%) to rats for periods up to 20 months showed no evidence of neoplastic changes.

Mutation studies of quinine (dihydrochloride) in male and female mice gave negative results by the micronucleus test. Intraperitoneal injections (0.5 mM/kg) were given twice, 24 hours apart. Direct *Salmonella typhimurium* tests were negative, when mammalian liver homogenate was added, positive results were found.

No information relating to the effect of quinine upon fertility in animal or in man has been found.

Pregnancy

Category X. See CONTRAINDICATIONS.

Nonteratogenic Effects

Because quinine crosses the placenta in humans, the potential for fetal effects is present. Stillbirths in mothers taking quinine have been reported in which no obvious cause for the fetal deaths was shown. Quinine in toxic amounts has been associated with abortion. Whether this action is always due to direct effect on the uterus is questionable.

Nursing Mothers

Caution should be exercised when Quinamm is given to nursing women because quinine is excreted in breast milk (in small amounts).

ADVERSE REACTIONS

The following adverse reactions have been reported with Quinamm in therapeutic or excessive dosage. (Individual or multiple symptoms may represent cinchonism or hypersensitivity.)

Hematologic: acute hemolysis, thrombocytopenic purpura, agranulocytosis, hypoprothrombinemia.

CNS: visual disturbances including blurred vision with scotomata, photophobia, diplopia, diminished visual fields, and disturbed color vision; tinnitus; deafness; vertigo; headache; nausea; vomiting; fever; apprehension; restlessness; confusion; and syncope.

Dermatologic/allergic: cutaneous rashes (urticarial, the most frequent type of allergic reaction, papular, or scarlatiniform), pruritus, flushing of the skin, sweating, occasional edema of the face.

Respiratory: asthmatic symptoms.

Cardiovascular: anginal symptoms.

Gastrointestinal: nausea and vomiting (may be CNS-related); epigastric pain.

DRUG ABUSE AND DEPENDENCE

Tolerance, abuse, or dependence with Quinamm has not been reported.

OVERDOSAGE

See prescribing information for a discussion on symptoms and treatment of overdose.

DOSSAGE AND ADMINISTRATION

1 tablet upon retiring. If needed, 2 tablets may be taken nightly—1 following the evening meal and 1 upon retiring.

After several consecutive nights in which recumbency leg cramps do not occur, Quinamm may be discontinued in order to determine whether continued therapy is needed.

Product Information as of October 1980

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Direct Medical Inquiries to

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Family Violence—Child Abuse And Neglect

Charles L. Pelton, M. D.*

"If you wish to give birth to a citizen and do without parental love, then be so kind as to warn society that you wish to play such an underhanded trick. People brought up without parental love are often deformed people" A. S. Makeenko

ABSTRACT

This paper presents the experiences and thoughts of a Family Practitioner concerning family violence with particular emphasis on child abuse and neglect. The psycho-social, emotional and physical aspects of this disease are discussed as well as suggestions for ways in which physicians can contend with violence in families. As one of the major "dys-eases" in our society, the physical, mental and verbal forms

of abuse and neglect must no longer be rationalized and ignored. The rigid adherence to traditional sex roles tends to foster household violence which is a symptom of a family in crisis. Today's physician has a moral, legal and social responsibility to assist in identification, prevention and treatment of this all to pervasive family event. Advocates for individual members of the family as well as the family as a unit are needed and there is no better place to begin than in the office of every physician.

Nearly two million children under the age of seventeen are abused each year by one of the parents and according to the National Institute of Mental Health almost 4% of the nation's children and adolescents are the subject of some form of child abuse. There appears to be an increase in family violence in this country and if we had two million children suffering from a communicable disease, it would be considered a major national epidemic.

Physicians in daily contact with families and children are in a strategic position to detect signs and symptoms of a dysfunctional family. This position allows the physician to assist in preventing abuse and neglect and to detect it in the early stages. We must know what the keys are and receive training

in the area of family violence while learning what child abuse and neglect encompasses. We must know what can be done for the victim, the perpetrator and the family. There must be a willingness to look for role-reversal and symbiosis in a family. What is the meaning of a child's becoming withdrawn or depressed and having sleeping or eating disturbances? Furthermore, we must know how to give appropriate services for those in need, know what community resources exist and what specialists are available in treating these types of families. Knowing the reporting laws governing child abuse and neglect and what agencies are available to protect the child and the family is imperative.

Each community needs a coordinated team to adequately deal with this form of human violence. This team must be equipped to formulate ideas, educate the public, share ideas across inter-agency lines to ensure the child is protected and the family receives the needed treatment. Whenever court ac-

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tion is necessary it must be recommended.

We in our culture are finding it increasingly difficult to bear the pain of our children. We no longer have the excuse we used to have when people had large families and could say they were too busy to give each child individual attention. So we rationalize our collective neglect of children by denying that the neglect does them any harm. Just as each of us has individually repressed all painful memories of those earliest most vulnerable years, we would like to be able to say of children in general: **early experience does not matter, it all just goes away and is forgotten.**

Among the disturbing findings in the National Institute of Mental Health's report was that parents under the age of thirty are 60% more likely to beat a child than are parents between the ages of thirty-one and fifty. A national comprehensive survey involving 1,100 families nationwide indicates the type of parent most likely to beat a child:

1. Black parents and white parents are equally likely to beat their children, but beatings by minorities are more likely to be reported to authorities.
2. The south has the lowest incidence of child beating, 10%. The midwest has the highest, 19%.
3. Poor parents are much more likely to beat their children than middle-income or wealthy parents.
4. Parents in large cities tended to abuse children more than parents in small cities, suburbs or rural areas.
5. Unemployed parents are 60% more likely to abuse their children than are employed parents.
6. Blue collar parents are 45% more likely to abuse their children than are white collar parents.
7. Men and women are equally likely to abuse their children. But in families that are heavily dominated by either parent, child abuse is much more likely.
8. Parents who are highschool graduates had a higher percentage of abuse than did either college graduates or parents with no more than an eight grade education.
9. Only 6% of Jewish parents surveyed had abused children, compared to almost 15% of Catholics and Protestants.

Who then are child abusers? They are the people with a missed childhood, a disastrous upbringing which causes serious developmental gaps. They are isolated and have learned not to trust. These people have a strange perception that children should give them the love and affection they never got during their own childhood. These care-takers of children

often display a flat affect with a lack of impulse controls and exhibit considerable denial. Obstruction toward discovery and treatment with inappropriate under or over-reaction to the stresses of treatment are seen. All socio-economic levels are included.

The child's behavior can be a tip-off to abuse: inappropriate clinging, total compliance, frozen watchfulness, fearfulness, depression and withdrawal are often the danger signals.

Any "special" child is at risk for abuse and include: the malformed, those with hearing or visual difficulties, colic and hyperactivity. The premature and the exceptionally intelligent or those who do not appear to fit into the mold of society are also at risk. Any child that does not have the opportunity to experience positive bonding with the parents is at high risk. (This is one reason I encourage breast feeding.)

One of the real dangers of child abuse is that frequent parental abuse increases the possibility that a boy will become a woman beater and that a girl will become a woman who will resignedly suffer abuse.

Rigid adherence to traditional sex roles also tends to foster household violence, encouraging the man to physically assert his dominance and the woman to submissively bear the blows. In violent households, women have a tendency to find themselves in accordance with the man's expectations. These expectations include her willingness to submit because they believe themselves to be totally responsible for the family's emotional climate. They view their ability to tolerate violence and hold the family together as a sign of strength.

Family stress caused by economic difficulty and the need for both partners to work contributes to violence, particularly if the male feels inadequate as breadwinner. This stress is developed into "dysstress" because of this parental immaturity, instability, volatility, anti-social behavior and poor impulse control. The parents themselves are usually socially isolated, experiencing financial difficulty, exhibiting signs of depression and may be maintaining a poor employment record. Furthermore, abusing parents were themselves frequently abused children and like many parents are visiting past experiences on their own children.

There is a fine line between disciplining, over-discipline and abuse with there being an increased risk of child abuse and neglect in homes where physical disciplining is the norm. Many of these parents learned violent assaultive forms of disciplining from their parents and have not learned how to deal with children in a nonassaultive manner. Furthermore, couples with a tendency for violence are more apt

to neglect and physically or emotionally abuse their children.

There are several fallacies that preclude a complete understanding of child abuse and the parents who perpetrate that abuse:

1. Maternal instinct is an illusion and it is a myth to assume that a biological mother is endowed with an innate positive feeling for her infant or child.
2. Contrary to many reports, psychosis is rarely a factor in child abuse. Rather the abusing parent may suffer from a personality or character disorder and likely to be maladjusted and unable to handle stress. The majority of parents are sane and aware of what they are doing with some of them justifying their violence and others feeling profound guilt.
3. Human violence and aggression are not instinctive behaviors. They are learned and rooted in cultural determined practices which are condoned by social values and incited by life's stress.
4. A parent is not a potential child abuser whose violence can be unleashed by sufficient provocation. Non-abusive parents have normal mental mechanisms which prevent them from harming their children. Severely abusive parents usually have poor ego control, lack of self-restraint and inability to place life's frustrations in the proper perspective.

Child abuse and neglect is a symptom of a family in crisis. Not only the abused child and the abusing parent must be involved in counseling and therapy but also the spouse and other siblings. Abuse of alcohol and drugs is frequently seen as these agents are used to escape frustration, responsibilities and daily stress.

Those of us living in industrialized nations presume to constitute a civilized society. However, one aspect of our social development, the protection of children from neglect and abuse has shown a marked lack of progress until the very recent past. Child abuse is only now becoming a major issue of concern.

The full responsibility for the physical, emotional, mental and sexual well-being of children has historically been placed upon parents, whether or not they were capable of providing proper care. In the past, parents who abused their children did so with little interference from society. Today, however, attempts are being made to provide medical care, social services and other aid to those individuals who are totally dependent upon responsible adults for their survival.

Child abuse has existed for centuries with varying degrees of acceptance by society. In earlier times, the destruction of children considered undesirable

for religious, political or economic reasons, or just because they were defective or female, was often culturally approved. In Biblical times, Herod destroyed an entire sector of male infants in his district. During the 14th century unwanted babies were thrown into the Thames without significant interference by society; during the Industrial Revolution young children were beaten and forced to perform dangerous and unhealthy tasks for long hours in filth ridden environments; and throughout history young boys and girls have been sold or kept for sexual purposes. Literature abounds with characterizations of children subjected to abuse. The most notably, the works of Dickens, Hardy and the Brothers Grim vividly portray exploitation of children by adults.

The first legal interference on behalf of an abused child occurred in the United States in 1874. Ironically, the outcry was raised by the American Society for Prevention of Cruelty to Animals. The child involved in this case was malnourished and regularly beaten by her adopted parents and she obtained legal protection on the basis that she belonged to the Animal Kingdom. Not until the 1960s and the 1970s did most states adopt laws requiring reporting of incidents of child abuse and providing immunity for those who filed the report if charges are made in good faith.

The phrase "the battered-child syndrome" was coined in 1962 by C. Henry Kempe, M.D. and was referred to in the first comprehensive survey concerning the identification and incidence of child abuse. Florid cases are easily recognizable, but mild to borderline cases present the most diagnostic difficulties. These borderline cases, therefore, are of greatest concern because the failure to identify an incident of abuse precludes protection for the child and support for the parents.

A broad spectrum of abuse and neglect situations exists. Some forms are easily recognized and are treatable; others, such as emotional abuse are only now being defined and recognized; still others, such as the effects of oppressive poverty or child prostitution, defy description and management. As in the case of most other illnesses, awareness and prevention of abuse promises to be more productive than treatment after the disease is flourishing.

Gil contends that child abuse takes place on three separate, though interrelated, levels.

Level One is abuse restricted to the individual child and family.

Level Two is institutional abuse (involving individual child or children as a group) taking place in schools, courts, foster care homes, or health and welfare delivery systems.

Level Three is abuse on the social level, including

legislation dangerous to, or negligent of, the needs of children or families.

Furthermore, he contends that management programs for child abuse must encompass all three levels before they can be successful.

Although there are many different forms of abuse and neglect, they may exist as single entities or occur in combination. The clinical finding of a fractured radius does not preclude the possibility that the child has been neglected or emotionally abused.

The basic definition of physical abuse is the non-accidental injury of a child. Injury may be the result of a single episode of abuse or may occur repeatedly and can range in severity from minor to fatal.

Physical punishment is widely accepted and commonplace in our society. Any injury that requires medical treatment is outside the range of normal corrective measures. In addition, any punishment that involves hitting with a closed fist or an instrument, kicking, inflicting burns, or throwing the child obviously represents child abuse regardless of the severity of the injury sustained.

Physical neglect is defined as failure to provide the necessities of life for a child. The lack of medical care, adequate nourishment, appropriate clothing, supervision and adequate housing all are factors that constitute neglect. However, great care must be taken in diagnosing cases of willful neglect; impoverished families may be providing the best care possible within their means.

Sexual abuse refers to any sexual activity between an adult and a child. Sexual abuse can be either assaultive or non-assaultive. It is probably the least reported form of child abuse because it is often non-assaultive and the abuser is a parent of the child with family members not likely to reveal the abuse.

Emotional abuse and neglect are extremely difficult to define and manage. These forms of abuse are committed by those parents who fail to provide a loving environment in which their child can thrive, learn and develop. Such failure may be manifested by ignoring, threatening, terrorizing or blatantly rejecting the child.

The effects of emotional (or psychosocial) abuse on the child include failure to thrive and various learning problems. Diagnosis is difficult because these effects are not as dramatic as bruises and lacerations. As a result, obtaining court required assistance for families involved in this form of abuse is much more difficult than in other cases. Compounding the problem of documentation of emotional abuse are the cultural variations and expectations concerning the roles of parents and children; behavior that constitutes emotional abuse in one social group may be acceptable in another.

Child abuse is a family affair and may occur in

the form of an isolated incident or may be chronic. A prerequisite for any single episode of abuse is a child who is difficult to manage (or is merely considered difficult by the parents), a parent or family that has the potential to abuse, and a stressful event that precipitates the abuse.

Multiple episodes of physical, sexual or emotional abuse may have the same characteristics as a single episode of abuse; but a single, clearly defined crisis event is generally lacking. In families involved in chronic emotional abuse, the situation is frequently compounded by significant emotional disturbances in one or more of the family members.

In order to aid the physician in making prompt and accurate identification of the abused child, a comprehensive description of characteristics shared by the types of events that may precipitate an episode of abuse must be understood.

Many parents abuse their children because of their immaturity and insecurity with a lack of understanding of children's needs and behavior. There are often unrealistic expectations where parents expect children to behave "like adults" at all stages of development. Unmet emotional needs are prevalent where parents do not relate well to other adults and expect children to take care of them, satisfy their needs for love, protection, and self-esteem. There is also frequent crises where financial, job, legal problems, major illnesses and other entities trigger a parent to "take it out" on a child. The lack of "parenting" knowledge where parents do not know the various stages of child development or how to raise a child are major issues. Furthermore, the parents themselves did not have adequate role modeling of successful family relationships from which to learn.

Social isolation with few friends or family to assist with the heavy demands of small children along with poor childhood experiences and the development of poor self-image create a potentially abusive environment.

Although relatively few parents are criminal or mentally unbalanced, every parent has the potential to abuse or neglect a child at some time, and most abusive and neglectful parents are "normal."

Even though sexual molestation of children occurs as often as 360,000 times per year in the United States, and probably more often than that, this form of abuse is the least-often reported because of social taboos regarding sex. Sexually abused children may present with injuries to the genitalia or rectum but most frequently the suspicion of sexual, as well as other forms of abuse, is raised by listening to what the child has to say and by observation of that child's behavior. Vague complaints of abdominal pain, sleep disturbances, or of personality changes should

alert one to search for an underlying source of complaints of sexual child abuse.

The child's father is frequently the offender and the abuse occurs repeatedly. This is a difficult entity to define, but of the categories of sex crimes committed against children, rape and attempted rape account for the greatest percentage. Carnal abuse, which is generally defined as any indecent or immoral practice involving the genitalia of a child is the next most common with sodomy being next in frequency and incest being the least reported. Often the child is coerced by direct force or by threat of bodily harm. These children are often victimized by relatives or friends who abuse the child's loyalty and affection. Many of the incidents occur in the family home, often while other people are in the home. Most of the reported cases of sexually abused children are girls with the median age being eleven years.

It is not enough to tell a child not to take candy or accept bribes from a stranger, as 75% of child molesters are people the child knows. The most important point to impress upon children is that they are not obliged to accept any form of physical affection that makes them feel uncomfortable.

The most helpful service that health professionals can provide is to be receptive to these children. Enormous physical, emotional and mental damage can result from sexual assault. These children, as do all children, require careful gentle evaluation and treatment.

Undoubtedly one of the most significant areas of sexual neglect involves the area of formal sex education. In order to prevent the susceptibility of children to sexual abuse, we as a society, must provide adequate and accurate sex education. I am of the opinion that this education must be started in the home and continued through the years of formal institutional education.

One of the prerequisites to getting help for children and to launching campaigns on their behalf is recognition of the rights of children. The law still emphasizes the rights of parents. The child is not guaranteed—nor does that child have an absolute right to—parental care that will provide adequate food, clothing and shelter. Children do not have rights to medical and health care that insure a healthy future. They do not have rights to a safe home and healthy environment. Furthermore, they do not even have a right to their own body.

Almost a decade ago, the Joint Commission on Mental Health of Children stated . . . "children have the right to be wanted, to live in a healthy environment, to have their basic needs satisfied, and to receive loving care." Yet, such rights are still not recognized. Therefore, laws must be changed

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ANUSOL-HC® SUPPOSITORIES

Hemorrhoidal Suppositories with Hydrocortisone Acetate

ANUSOL-HC® CREAM

Rectal Cream with Hydrocortisone Acetate

Caution: Federal law prohibits dispensing without prescription.

Description: Each Anusol-HC Suppository contains hydrocortisone acetate, 10.0 mg; bismuth subgallate, 2.25%; bismuth resorcin compound, 1.75%; benzyl benzoate, 1.2%; Peruvian balsam, 1.8%; zinc oxide, 11.0%; also contains the following inactive ingredients: dibasic calcium phosphate, and certified coloring in a hydrogenated vegetable oil base.

Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg; bismuth subgallate, 22.5 mg; bismuth resorcin compound, 17.5 mg; benzyl benzoate, 12.0 mg; Peruvian balsam, 18.0 mg; zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water.

Anusol-HC Suppositories and Anusol-HC Cream help to relieve pain, itching and discomfort arising from irritated anorectal tissues. These preparations have a soothing, lubricant action on mucous membranes, and the antiinflammatory action of hydrocortisone acetate in Anusol-HC helps to reduce hyperemia and swelling.

The hydrocortisone acetate in Anusol-HC is primarily effective because of its antiinflammatory, antipruritic and vasoconstrictive actions.

Indications and Usage: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain, itching and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas, pruritus ani and relief of local pain and discomfort following anorectal surgery.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

Contraindications: Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

Precautions: General: Symptomatic relief should not delay definitive diagnoses or treatment.

Prolonged or excessive use of corticosteroids might produce systemic effects.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

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Anusol-HC is not for ophthalmic use.

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and public attitudes modified.

The sacredness of the home is used by many in an attempt to prevent those around from knowing what is going on within. If we are going to continue this attitude we must provide people with the knowledge and training to become responsible, loving, caring and understanding parents. Because self-esteem is the most important word in family violence, it is critical that the parents learn how to meet their wants and needs while learning how to fulfill the needs of their children. Parents who have not learned how to manage stress or are under "dystress" are usually lonely and isolated; they must be encouraged to have contact with the outside world but also ensure their children have similar contact with their own peers.

"You can't take an x-ray of a shattered self-concept," said Dessie, but unless our society learns how to operate on a non-crisis basis our self-images will continue to suffer. We must learn how to respond to every day changes in life from a less anxious standpoint.

From the vantage point of a family physician, it is apparent that many changes can and must be made in dealing with family violence in our culture. Physicians working with other social and health care providers can have a great impact on this disease. By increasing public and professional awareness much can be done to insure a healthy individual, a wholesome society and a safe environment in which to work and play.

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Future Meetings

December

American Cancer Society National Conference-Gastrointestinal Cancer 1981, Fontainebleau Hilton, Miami Beach, FL, Dec. 8-10. 13 hrs. AAFP and AMA Category I credits. Contact: Nat'l. Conference on Gastrointestinal Cancer, 777 3rd Ave., New York, NY 10017.

January

Ophthalmology Clinical Conference, Univ. of Iowa Hosp. and Clinics, Jan. 6. AMA Category I credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa College of Medicine, Iowa City, IA 52242.

Advanced Trauma Management, Univ. of Iowa Hosp. and Clinics, Jan. 8-9. AMA Category I credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa College of Medicine, Iowa City, IA 52242.

Advanced Cardiac Life Support, Univ. of Iowa Hosp. and Clinics, Jan. 11. AMA Category I credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa College of Medicine, Iowa City, IA 52242.

Supercourse VII, Fairmont Hotel, New Orleans, LA, Jan. 13-16. Fee: \$225. Category I credits. Contact: Course Coordinator, Am. Lung Assoc. of LA, 333 St. Charles Ave., Suite 500, New Orleans, LA 70130.

Program For Chiefs Of Clinical Services, School of Public Health, Boston, Mass., Jan. 17-30. Fee: \$2700. AMA Category I credits. Contact: Ass't. Director for Administration, Exec. Programs in Health Policy, Harvard School of Public Health, 677 Huntington Ave., Boston, MA 02115. Phone: (617) 732-1142.

Mayo Foundation Outreach Seminar—Dermatology, McKennan Hosp. Aud., Sioux Falls, SD, Jan. 15. 6 hrs. AAFP and AMA Category I credits. Contact: Ruth A. Muchow, Ed. Center Coordinator, McKennan Hosp., 800 E. 21st, Sioux Falls, SD 57105. Phone: (605) 339-8000.

Advanced Cardiac Life Support, Univ. of Iowa Hosp. and Clinics, Jan. 13. AMA Category I credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa College of Medicine, Iowa City, IA 52242.

Radiology For The Non-Radiologist, Innisbrook, FL, Jan. 18-22. 25 hrs. AMA Category I credits. Contact: Edward A. Eikman, M.D., Assoc. Prof. of Medicine, U. of South Florida Coll. of Medicine, V. A. Hosp., 1300 N. 30th St., Tampa, FL 33612. Phone: (813) 974-2032.

Radiation Therapy Seminar, Univ. of Iowa Hosp. and Clinics, Jan. 21. AMA Category I credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa College of Medicine, Iowa City, IA 52242.

February

Cardiac Dilemmas, Univ. of Iowa Hosp. and Clinics, Feb. 3-4. AMA Category I credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa College of Medicine, Iowa City, IA 52242.

Ophthalmology Clinical Conference, Univ. of Iowa Hosp. and Clinics, Feb. 4. AMA Category I credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa College of Medicine, Iowa City, IA 52242.

Mayo Foundation Outreach Seminar—Neurology-Acute Trauma, McKennan Hosp. Aud., Sioux Falls, SD, Feb. 12. 6 hrs. AAFP and AMA Category I credits. Contact: Ruth A. Muchow, Ed. Center Coordinator, McKennan Hosp., 800 E. 21st, Sioux Falls, SD 57105. Phone: (605) 339-8000.

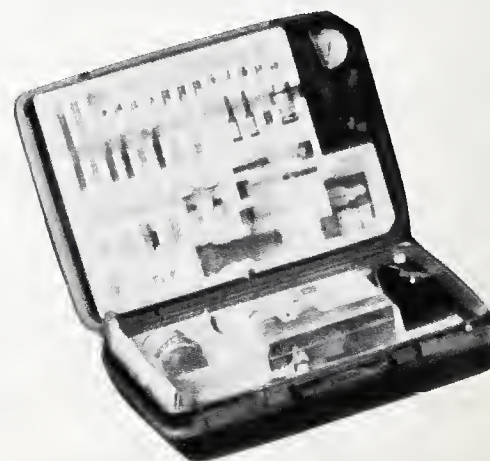
Advanced Cardiac Life Support, Univ. of Iowa Hosp. and Clinics, Feb. 12-14. AMA Category I credits. Contact: Richard M. Caplan, M.D., Assoc. Dean for CME, Univ. of Iowa College of Medicine, Iowa City IA 52242.

Conference On Genetic Counseling, The Church And The Law, Airport Holiday Inn, Sioux Falls, SD, Feb. 13-14. Contact: Robert H. Quinn, M.D., Prof. Div. of Surg., USDSM, 2501 W. 22nd, Sioux Falls, SD 57105. Phone: (605) 339-6791.

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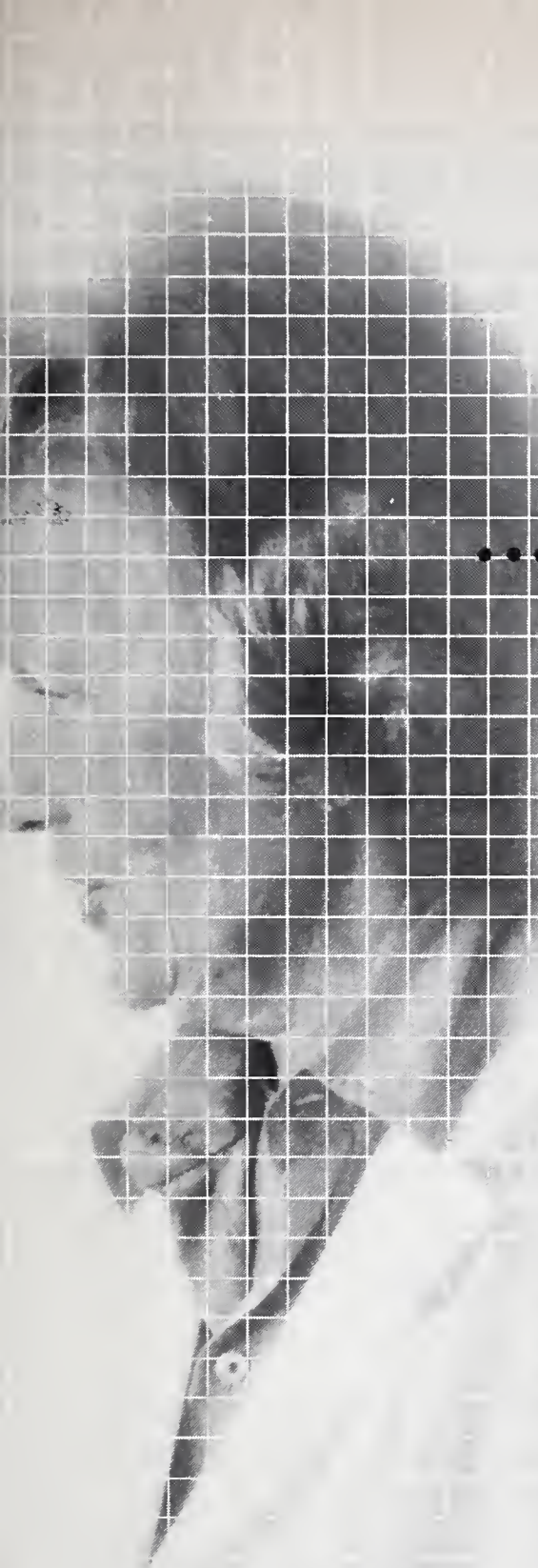


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Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication, abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed, drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation. The clearance of Valium and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported, should these occur, discontinue drug. Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. **Adults:** Anxiety disorders, symptoms of anxiety, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed, adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d., adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. **Geriatric or debilitated patients:** 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) **Children:** 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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NEXT MONTH

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Fifty-Seven Year Old Caucasian Male
With Anemia Of Thirty Years Duration



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and
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**SOUTH DAKOTA
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An Approach To Diagnosis And Management Of Thyroid Nodules

F. Lovrien, M.D.*
Discusser

J. F. Barlow, M.D.**
Editor

DR. JOHN BARLOW: I would like to alter our usual proceedings and have Dr. Lovrien explain his approach to common problem—the thyroid nodule.

DR. FRED LOVRIEN: Today I would like to discuss the common problem of the thyroid nodule. The most controversial nodule is a solitary nodule which shows hypofunction on a radionuclide scan. There are several approaches to a solitary thyroid nodule. To avoid missing a malignancy, some recommend surgery be performed in all cases. A diametrically opposite approach is to suppress all thyroid nodules with thyroid hormone, and operation is reserved for only those nodules which fail to shrink. I feel a better approach is intermediate between these two strategies. Logical use of the history, physical examination, laboratory and other ancillary tests will result in the most benefit with the least risk to an individual patient.

I. Statistical approach to thyroid nodules.

There have been multiple studies on the prevalence of thyroid nodules. In the Framingham study,

investigators palpated the thyroid gland and found 3% of patients with palpable solitary thyroid nodules and 1% with multiple nodules.¹ Half of the palpable solitary nodules were multiple nodules at autopsy.² In 1959 Sokal also found a 4% prevalence of thyroid nodules in a large series of North American patients.³ With this high prevalence of thyroid nodules, one would predict over 8 million patients in the United States would have palpable thyroid nodules. If these nodules were commonly malignant, one would expect a significant morbidity and mortality. However, the U.S. Public Health Service shows the incidence of thyroid carcinoma in the United States is 25 per million population per year. The fatality rate is only 5 per million for men and 8 per million for women.⁵ The high incidence and low fatality rate of thyroid nodules suggests most thyroid nodules have an extremely low mortality rate.

The prognosis of most thyroid cancers is even less ominous when one considers the frequency of asymptomatic, microscopic cancers at autopsy. In the United States, most autopsy studies show a 2-4.2% prevalence of microscopic carcinoma of the thyroid in unselected patients.⁶ Multinodular goiters are estimated to be 13% or even higher.^{7,8} Whether these microscopic carcinomas frequently progress and lead to morbidity and mortality is highly controversial. Many feel the majority of microscopic

* Specialist in Endocrinology and Medicine, Central Plains Clinic and Sioux Valley Hospital; Clinical Associate Professor of Medicine, School of Medicine, University of South Dakota.

**Pathologist, Laboratory of Clinical Medicine and Sioux Valley Hospital; Professor of Pathology, School of Medicine, University of South Dakota.

cancers would remain clinically silent without adverse effects indefinitely.

II. Clinical approach to thyroid nodules.

Our goals are to determine which thyroid nodules are malignant and to determine the type of thyroid malignancy. There are several important historical factors and physical findings which affect the likelihood of malignancy in a given nodule:

A. History.

1. **Sex:** A given thyroid nodule has a greater chance of malignancy in males than in females. However, non-malignant thyroid diseases are several times more common in females. Therefore, in an individual patient, a thyroid abnormality is more likely benign in females.
2. **Age:** A thyroid nodule in a patient over 40 years of age is less likely malignant than a nodule in a patient under forty years of age.
3. **History of head or neck irradiation:** Dr. Leslie DeGroot was one of the first physicians to draw attention to the association between head and neck irradiation in childhood and papillary carcinoma of the thyroid.⁹ Generally, the irradiation had been for an enlarged thymus, tonsils, acne, etc. This association has subsequently been questioned, although most feel a prior history of head or neck irradiation increases the risk of papillary carcinoma.
4. **Family history:** A positive family history for thyroid cancer slightly increases a given patient's risk of malignancy. This is especially true if the family history is positive for medullary carcinoma, often part of a multiple endocrine neoplasia syndrome.
5. **Hoarseness:** A history of recent hoarseness increases the risk of malignancy in a thyroid nodule. Hoarseness can also be seen in thyroiditis and other conditions, but may be an ominous symptom.
6. **Recent increase in size:** A nodule which has shown progressive increase in size over a relatively short period of time is suspicious for a malignant nodule. Cystic degeneration or hemorrhage in a nodule or thyroiditis may less commonly present in this manner.

B. Physical examination.

1. **Number of nodules:** Estimates of malignancy in a multinodular goiter range from 1-20%.^{7,8} The incidence of malignancy in a solitary cold nodule is generally estimated at 10-20%.¹⁰ Although there is a great overlap, it is agreed that the frequency of malignancy is greater in solitary nodules than

in multiple thyroid nodules.

2. **Consistency of the nodule:** A hard nodule has nearly a 45% chance of malignancy. The risk of malignancy in a soft nodule ranges from 8-11%. In experienced hands, consistency is a better predictor of malignancy than is function on a radionuclide scan.
3. **Signs of local pressure:** hoarseness, stridor or damage to the recurrent laryngeal nerve often indicate a malignant process.
4. **Regional lymphadenopathy:** Although thyroiditis may also result in lymphadenopathy, malignancy produces regional lymphadenopathy in a much higher percentage of patients than do benign processes.
5. **Transillumination:** Occasionally a large cyst can be easily transilluminated, indicating a low likelihood of malignancy.

C. Laboratory testing.

Laboratory testing is not often helpful in predicting malignancy in a thyroid nodule. Most patients with solitary nodules are euthyroid. However, there are several circumstances in which laboratory evaluation may be helpful.

1. In all patients a T-4 is helpful for initial classification of nodules. (See Diagram I)
 - a. If the patient is clinically hypothyroid, or if the T-4 is low normal or low, a serum TSH is indicated. A low T-4 with an elevated serum TSH confirms primary hypothyroidism.
 - b. If the patient is clinically hyperthyroid, a serum T-4 may confirm this. If the T-4 is normal, a T-3 by RIA is indicated. There is an increased frequency of T-3 thyrotoxicosis in autonomous nodules.
 - c. In most cases the patient will be clinically euthyroid and the T-4 will be in the normal range.
2. If the patient has a family history of thyroid malignancy, or symptoms of flushing, sweating, diarrhea, or other symptoms suggesting a medullary carcinoma are present, a serum calcitonin should be drawn. An elevated serum calcitonin is highly suggestive of a medullary carcinoma of the thyroid. Occasionally fasting serum calcitonin may be normal with medullary carcinoma. If suspicion is great, stimulation of serum calcitonin with pentagastrin or calcium may be necessary.¹¹
3. Antithyroid antibodies are rarely helpful. Very high levels of anti-thyroid antibodies are generally seen in only Hashimoto's thy-

roiditis or Grave's disease. However, low levels may be seen in multiple other conditions, including malignancy. Also, lymphoma of the thyroid gland often arises in a pre-existing goiter or Hashimoto's thyroiditis with elevated antibodies.

4. Serum thyroglobulin has been suggested as a useful test in the diagnosis of thyroid malignancy. However, its usefulness for this purpose is doubtful. In the future, it may become a useful marker in a patient with known thyroid malignancy. An increase in the serum thyroglobulin may predict recurrence.¹³ In addition to other problems, serum thyroglobulin levels are difficult to perform and are not commercially available at this time.

D. Radionuclide Scanning.

There are a variety of methods for scanning the thyroid. Today I will discuss only two—technetium 99 and I¹³¹. In most cases, the technetium scan should be used first. It involves less radiation to the patient, is more easily performed, is quicker, and is generally less expensive. However, the radiation is too weak to adequately visualize substernal extension. When a substernal goiter is suspected, I¹³¹ or I¹²³ are preferable tracers to use. The iodine scans are also preferred when localizing metastatic tumor in known thyroid malignancies. This can then be used in judging therapeutic efficacy with large dose I¹³¹. Lastly, an iodine scan may be used when a nodule appears functioning by technetium scanning.

Technetium is only trapped by follicular cells, but iodine must be organified in the process of thyroid hormone formation. Since the nodule must be more differentiated to use iodine, rarely a well differentiated malignancy may appear functioning with technetium but cold with I¹³¹.

The significance of warm or hot nodules vs. cold nodules continues to be controversial. A cold nodule does have a higher risk of malignancy. However, nearly 90% of cold nodules are still benign. Operating on all cold nodules results in an unacceptable incidence of surgery on benign disease.

Even the security of a functioning nodule on scan can be misleading. We have had two cases of proven malignancy in "warm" nodules over the last year. A scan has many similarities to an X-ray. The density on the scan is related not only to how actively tracer is taken up but

is also a function of the thickness of the nodule. A large nodule with some function may appear normal or even slightly hyperfunctioning. If the nodule is hot and suppresses the remainder of the gland, the chances of malignancy are negligible.

*DR. PHILLIP CARLSON. Were the hot nodules you described with iodine or technetium?

DR. LOVRIEN: They were with technetium.

E. Other Diagnostic Approaches.

Lastly, I would like to mention three other diagnostic modalities—soft tissue X-rays or xerography, ultrasound, and the thyroid needle aspiration biopsy.

1. X-rays

Soft tissue X-rays of the neck or xerography can be used to demonstrate calcification within a thyroid nodule. The multiple punctate calcifications of psammoma bodies are virtually pathognomonic of papillary carcinoma of the thyroid. Large areas of calcification may also occur in benign thyroid lesions, but psammoma bodies are relatively uncommon, and generally the cost-benefit ratio does not warrant their use.

2. Ultrasound.

Ultrasound is a diagnostic technique to differentiate cystic lesions from solid lesions.¹⁴ Cysts will generally show an echo free space. Solid lesions have internal echos and appear much like the surrounding thyroid tissue. If the lesion is solid, the ultrasound gives no further information on the risk of malignancy. With the advent of the thyroid needle aspiration biopsy, I feel the ultrasound no longer has a role in the approach to thyroid nodules.

3. Thyroid needle biopsy.

Several methods can be used to biopsy a thyroid nodule. A core biopsy can be obtained with a Vim-Silverman or a Tru-Cut needle. A safer technique involves using a skinny needle for aspiration biopsy by cytology.¹⁵ Today I will primarily address this latter technique. In 1978 two articles on thyroid needle aspiration biopsies were simultaneously published in the *Annals of Internal Medicine*:

- a. The first paper was by Dr. Gershengorn.¹⁶ He biopsied 27 thyroid nodules prior to surgical excision. Twenty-four of twenty-seven nodules showed a correct correlation between the aspiration biopsy and the excisional biopsy. In five cases, suspicious cells were seen. Two of these five cases proved to be malignant at surgery.

*Resident in Family Community Medicine.

Only one of twenty-seven cases showed a false negative diagnosis of malignancy in routine autopsy studies.

- b. The second study was published by Wal-fish et al.¹⁷ Eighty-three patients had thyroid aspiration biopsies after ultrasound examination. Sixty-three of sixty-six solid lesions (95%) were correctly diagnosed by the aspiration biopsy. Fifteen of seventeen (85%) of cystic or mixed lesions were correctly diagnosed preoperatively. This and other studies show the accuracy of diagnosis with aspiration biopsies in solid lesions is better than in cystic or partially cystic lesions.

Hamburger et al examined the problem from a different aspect.^{18,19} Rather than evaluating accuracy of diagnosis, they reviewed the effect of needle biopsies on how nodules were approached. He compared the approach in 455 nodules biopsied by the skinny needle aspiration technique with the approach used in 1,094 controls from previous years. The following changes were noted:

1. The patients with suspected cancer were halved.
2. The number of patients observed rather than subjected to surgery doubled.
3. The cases of thyroid carcinoma suspected and confirmed increased by 75%.
4. Operations on benign disease dropped 70%.

5. 42 of 47 cases with thyroid carcinoma were initially diagnosed by the skinny needle aspiration biopsy.

F. Summary.

In approaching a thyroid nodule, one must first determine its function and secondly determine its pathology. The general approach to these problems is outlined on Diagram I and Diagram II. Patients with nodules may initially be divided into hypothyroid, euthyroid, and hyperthyroid categories by thyroid function tests as described earlier in the discussion. (Diagram I)

1. In hyperthyroid patients, a scan will help differentiate an autonomous nodule with suppression of the remaining gland from a hypofunctioning nodule within a hyperthyroid gland. Surgery or radiation ablation may be necessary for larger or more symptomatic autonomous nodules. A solitary hypofunctioning nodule within a hyperthyroid gland should be biopsied.
2. If the patient is shown to have primary hypothyroidism with a low T-4 and an elevated serum TSH, 3-4 months on thyroid hormone suppression is indicated. If shrinkage occurs, conservative observation is adequate. If no shrinkage occurs, a needle biopsy or surgery is indicated.
3. The most difficult problem is a euthyroid patient with a solitary nodule. Diagram II outlines two philosophically different approaches. The first approach involves scan-

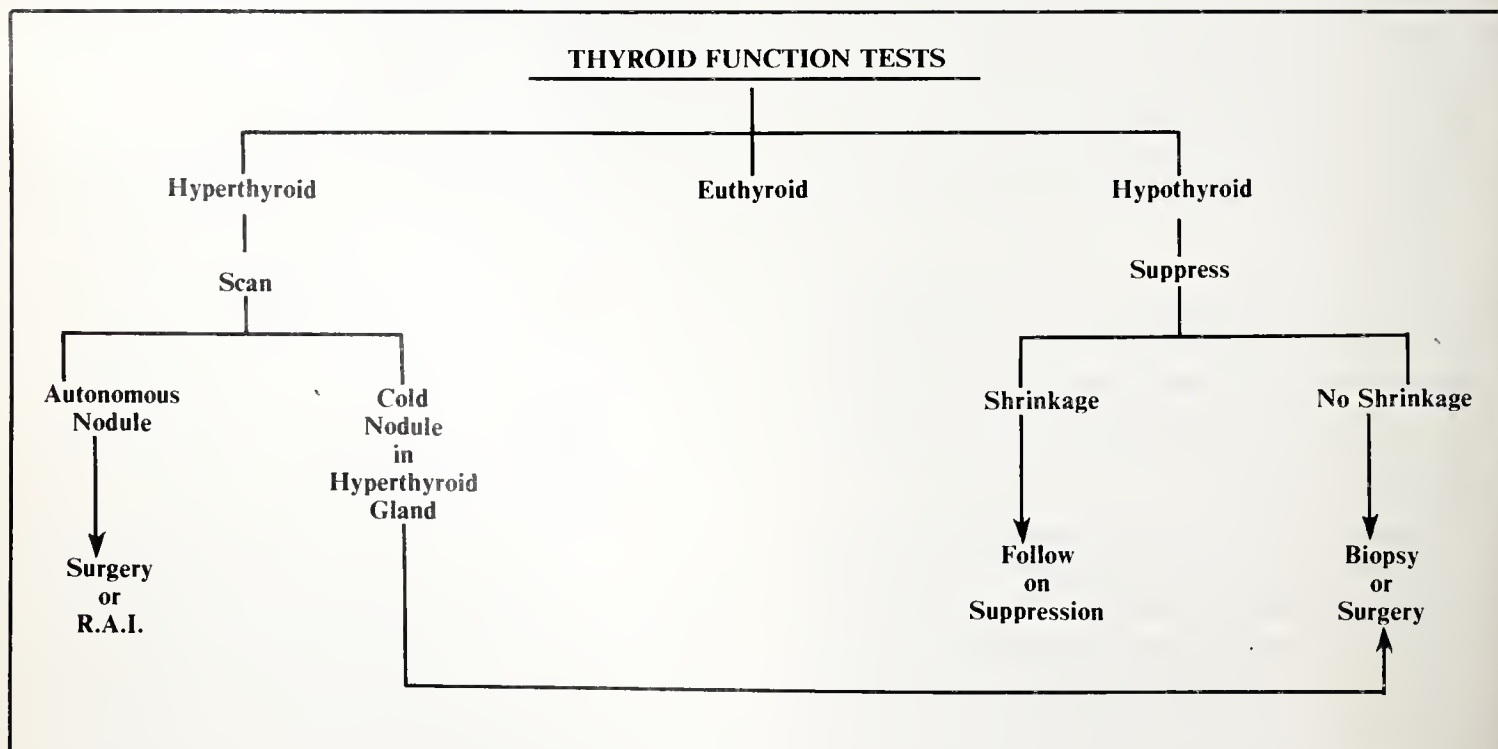


Diagram I

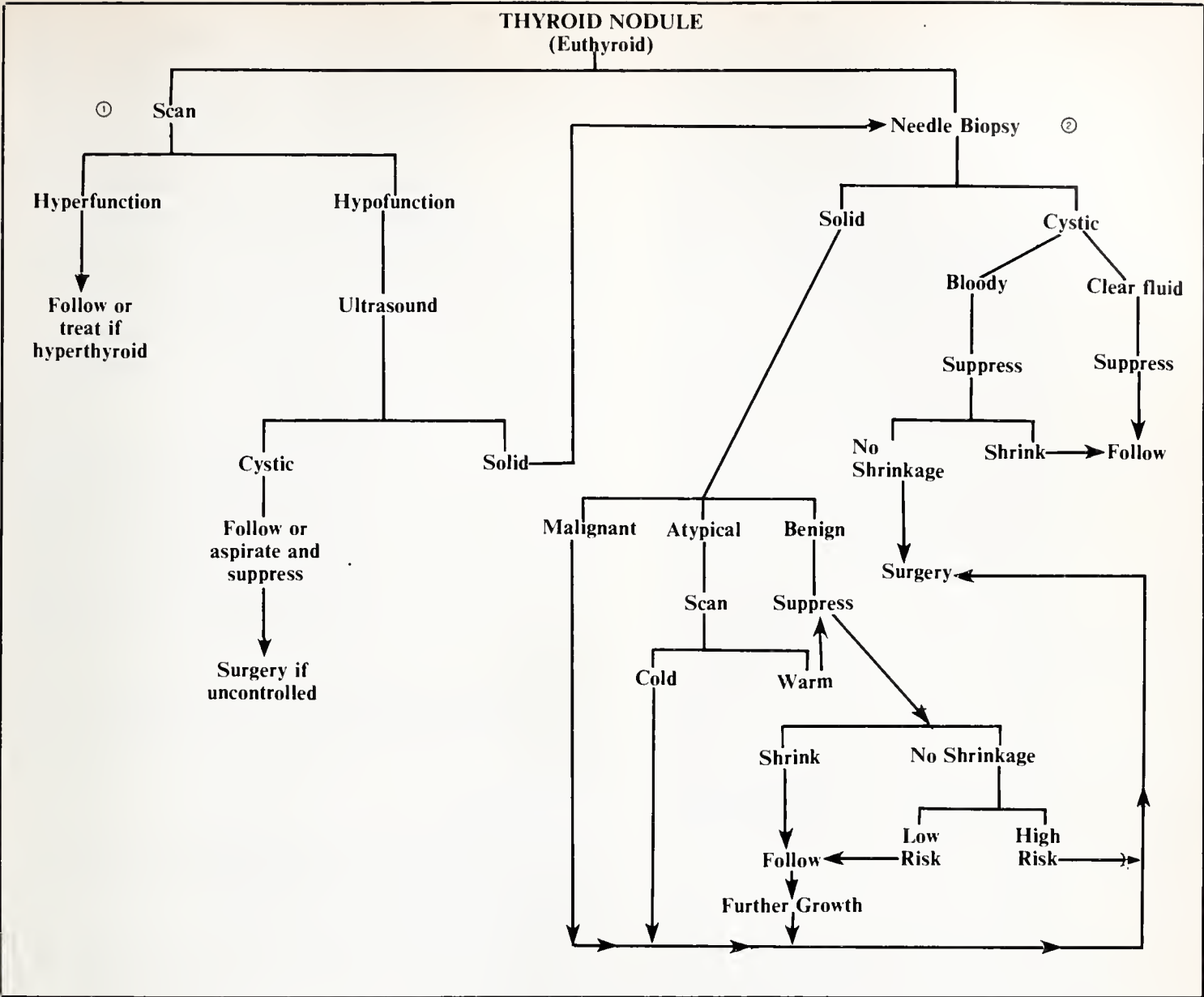


Diagram 11

ning as the initial procedure. The second approach involves the use of a needle biopsy as the initial step.

a. Currently the most commonly used approach involves a technetium scan as the first step. If the scan shows a functioning or hyperfunctioning nodule, this is generally followed or suppressed with L-thyroxine therapy. If the nodule is hypofunctioning, some proceed directly to surgery. This results in an unacceptably high incidence of surgery on benign disease. Some of these operations can be avoided with an ultrasound study to differentiate cystic from solid lesions. A cystic lesion may be aspirated, followed, suppressed with L-thyroxine, or a combination of these approaches may be used. If the cyst is uncontrollable with these measures, surgery is then used. If

the ultrasound study suggests a solid lesion, an aspiration biopsy or surgery is needed.

b. The newer approach involves the use of a thyroid skinny needle aspiration biopsy as the initial step in evaluation. If the lesion is cystic, the lesion is aspirated and cytologic examination of the fluid is done. Clear fluid almost certainly implies a benign lesion. A bloody fluid can be consistent with a benign or malignant process. These nodules are subsequently suppressed with L-thyroxine. If the nodule fails to shrink, surgery is necessary. If the needle biopsy shows a solid lesion, these can be categorized into malignant, atypical, and benign.

1. Malignant cytology is an indication for surgery.

2. Atypical cytology is an indication for a technetium scan. If the technetium scan shows the nodule to be functioning, a period of suppressive therapy with L-thyroxine is attempted. If the lesion suppresses adequately, suppression is continued indefinitely. If the nodule fails to suppress, surgery may later be necessary. If the scan is hypofunctioning, surgery is needed.
3. Benign cytology is an indication for L-thyroxine suppression. If the nodule shrinks over four months, this is followed indefinitely. If subsequent growth occurs, the nodule is surgically excised. If the lesion does not change in dimensions after suppression, one must decide, based on history and physical examination, if the patient is in a high or low risk category for malignancy. If the risk of missing malignancy is greater than the risk of surgery, surgery is the next step. If the risk of surgery is greater, permanent suppression is indicated. In any of these circumstances, persistent growth of the nodule despite adequate suppression is an indication for surgery.

Currently the use of a thyroid scan is the most commonly used approach. I feel this will change in the near future. I feel using the thyroid needle biopsy as the initial step has several advantages. Patients with cysts and malignancies are adequately diagnosed without either an ultrasound study or scan. The cytology is also more useful than a scan for determining which nodules can be safely suppressed.

There has been concern over seeding a needle track with malignant cells. This complication is extremely rare, and in those few cases in which it has occurred, the malignancy was metastatic to the thyroid or a primary anaplastic malignancy in the thyroid. The seeding of the needle tract also tended to occur with core biopsies.

I feel this newer approach is more accurate, more cost effective, and more

logical than using scans as the initial diagnostic test. In summary, we have a large number of diagnostic tests to help evaluate malignant potential in thyroid nodules. Logical and appropriate use of these tests will result in the greatest benefit with the least cost and risk to our patients.

*DR. JERRY SIELAFF. Is thyroid needle aspiration something you can do as an outpatient procedure, or do you admit the patient to the hospital.

DR. LOVRIEN: It is an outpatient procedure.

RESIDENT: You say you don't recommend ultrasound at this time?

DR. LOVRIEN: Not if the skinny needle aspiration biopsy is used as the first step. This obviates any useful information from the ultrasound. If one begins with a scan, it is possible that an ultrasound could give some useful information. However, I do not consider this the preferred approach. We have also had several cases in which a lesion was said to be solid and the needle biopsy showed the lesion to be cystic.

DR. SIELAFF: How many biopsies do you obtain?

DR. LOVRIEN: We do 5-6 passes through the nodule.

**DR. VAN ERT. Do you send the cyst fluid for cytology?

DR. LOVRIEN: If it is crystal clear serous fluid, I do not. If it is bloody, I will send this to be spun down for cytological examination.

DR. BARLOW: This is pretty much the way the surgeons handle the problem in aspiration of cystic breast lesions.

DR. CARLSON: What dose of L-thyroxine do not use?

DR. LOVRIEN: I use 0.15 mg. to 0.2 mg. of L-thyroxine per day.

***DR. RICHARD JAQUA. Do I understand that you treat a warm or functioning nodule as a cold nodule?

DR. LOVRIEN: The risk of malignancy in a warm or functioning nodule is definitely less than the risk of a hypofunctioning or cold nodule. However, I do not consider this a guarantee of benignity. We do have three documented cases over the last two years in which a "warm" or "functioning" nodule was shown to be malignant on skinny needle aspiration biopsy. I would place a functioning nodule on L-thyroxine suppression unless the nodule was autonomous. If the non-autonomous functioning nodule decreased in size on suppression, I would cautiously follow on long term suppressive therapy. If the nodule did not shrink, I would proceed with a skinny needle aspiration biopsy.

DR. BARLOW: The thin or skinny needle aspiration biopsy is not a new technique but has recently gained

* Resident in Family and Community Medicine.

** Resident in Family and Community Medicine.

*** Professor and Chairman, Department of Laboratory Medicine, University of South Dakota; Pathologist, Sioux Valley Hospital, Sioux Falls, SD.

increasing acceptance in this country. It has been used at Memorial Hospital in New York City since the 1920's and has been widely employed in Europe as an alternative method of making tissue diagnosis as compared to the more traumatic open biopsy techniques.

A 21-25 gauge needle attached to a suction syringe is advanced into a suspected lesion and the needle is moved back and forth in the lesion while negative pressure is applied by the syringe. Only a small amount of tissue or fluid, which may not even appear in the syringe, is obtained. The material should be immediately placed on slides and smears are made. In our technique, immediate fixation is required. Other techniques utilize air dried slides or placing the material in formalin for a cell block preparation. It is important to remember that gram or acid fast stain for microorganisms or Grocott stain for pneumocystitis carinii as well as a culture can also be performed under appropriate circumstances. This usually applies to lung aspirates. Disadvantages of the technique include small sample size and loss of tissue architectural patterns available with large biopsy specimens. With some experience and clinical correlation, definite diagnoses may be obtained. However, often equivocal or nondiagnostic cell spreads are observed. Only rarely has spread of tumor along the needle trace been reported.

Lesions of the thyroid, breast, lymph node, lung, retroperitoneal and pelvis may be approached by this technique. In some of the above locations, localization by computer tomogram scan, fluoroscopy, or ultrasound is necessary.

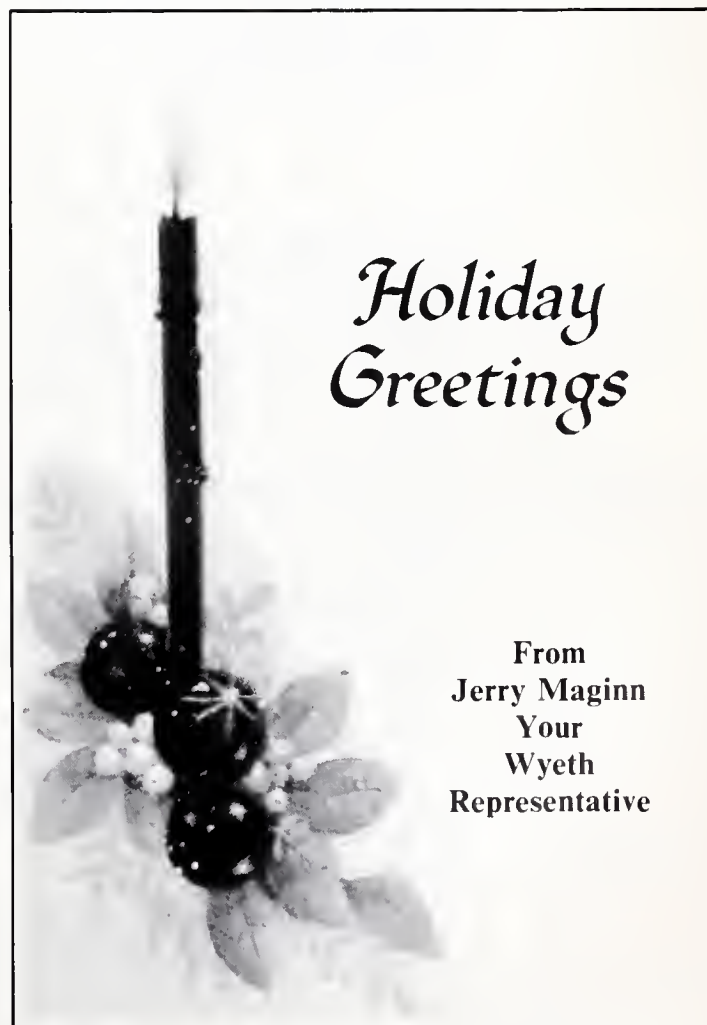
In selected cases major surgery may be obviated. It is important to realize that not every patient with a suspected lesion is a candidate and close cooperation between the clinician performing the biopsy and the pathologist is mandatory.

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Holiday Greetings

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WEDNESDAY, FEBRUARY 3, 1982

6:00 p.m. SDAFP Board of Directors Meeting—
Deer Mountain Room
7:00-9:00 p.m. Registration

THURSDAY, FEBRUARY 4, 1982

MORNING SESSION

Michael J. Brown, M.D., Moderator

7:00-7:30 a.m. Registration, Complimentary continental breakfast
7:30-8:10 a.m. "Cardiac Surgery—What To Expect and What Do I Tell Them"
Richard K. Parker, M.D.
8:15-8:55 a.m. "Real Time Two-Dimensional Echocardiography"
Michael A. Sarche, M.D.
9:00-9:40 a.m. "Long Term Results of Coronary Artery Bypass Surgery"
Richard K. Parker, M.D.
9:40-5:30 p.m. WINTER SPORTS TIME

EVENING SESSION

Raymond G. Nemer, M.D., Moderator

5:00-5:30 p.m. Registration, Complimentary coffee, hot wine and hot buttered rum
5:30-6:00 p.m. "Nuclear Cardiology Advances in Noninvasive Diagnosis, Part I—Static Imaging"
Michael A. Sarche, M.D.
6:05-6:35 p.m. "Use and Interpretation of Noninvasive Vascular Lab"
Richard K. Parker, M.D.
6:40-7:10 p.m. "Nuclear Cardiology Advances in Noninvasive Diagnosis, Part II—Gated Blood Pool Scanning"
Michael A. Sarche, M.D.
7:15-7:45 p.m. "Dialogue with Doctors"
AAFP Public Relations Program, Raymond G. Nemer, M.D. and Lawrence Finney, M.D., Board of Directors, SD Chapter, AAFP
7:45 p.m. EVENING FREE

FRIDAY, FEBRUARY 5, 1982

MORNING SESSION

Herbert Saloum, M.D., Moderator

7:00-7:30 a.m. Registration, Complimentary continental breakfast
7:30-8:10 a.m. "The Pharmacology of Calcium Blocking Agents"
James J. Scherrer, Pharm.D.
8:15-8:55 a.m. "Use of Calcium Blocking Agents in Vasospastic Angina"
James W. Jackson, M.D.
9:00-9:40 a.m. "Cardiac Pacing 1982"
Robert A. Van Tassel, M.D.
9:45-10:45 a.m. Optical Lab—"Use of Swan-Ganz Catheter"
Robert A. Van Tassel, M.D.
9:40-5:30 p.m. WINTER SPORTS TIME

EVENING SESSION

Lawrence Finney, M.D., Moderator

5:00-5:30 p.m. Registration, Complimentary coffee, hot wine and hot buttered rum
5:30-6:10 p.m. "New Anti-Arrhythmic Agents"
James W. Jackson, M.D.
6:15-6:55 p.m. "Drug Therapy in Hemodynamic Monitoring—Part I"
James J. Scherrer, Pharm.D.
7:00-7:40 p.m. "Comprehensive Evaluation and Management of Patient with Coronary Artery Disease"
Robert A. Van Tassel, M.D.
7:40 p.m. EVENING FREE

SATURDAY, FEBRUARY 6, 1982

MORNING SESSION

Charles L. Swanson, M.D., Moderator

7:00-7:30 a.m. Registration, Complimentary continental breakfast
7:30-8:10 a.m. "Recognition and Management of Complications of Acute MI"
Robert A. Van Tassel, M.D.
8:15-8:55 a.m. "Two New Agents for Hypertension—Captopril and Minoxidil"
James W. Jackson, M.D.
9:00-9:40 a.m. "Drug Therapy and Hemodynamic Monitoring—Part II"
James J. Scherrer, Pharm.D.
9:45-10:45 a.m. Optional Lab—"Uses of Temporary Pacer"
Robert A. Van Tassel, M.D.
9:40 a.m. Seminar Closes

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MEETING INFORMATION

NEXT ANNUAL MEETING—

ELECTION OF OFFICERS.

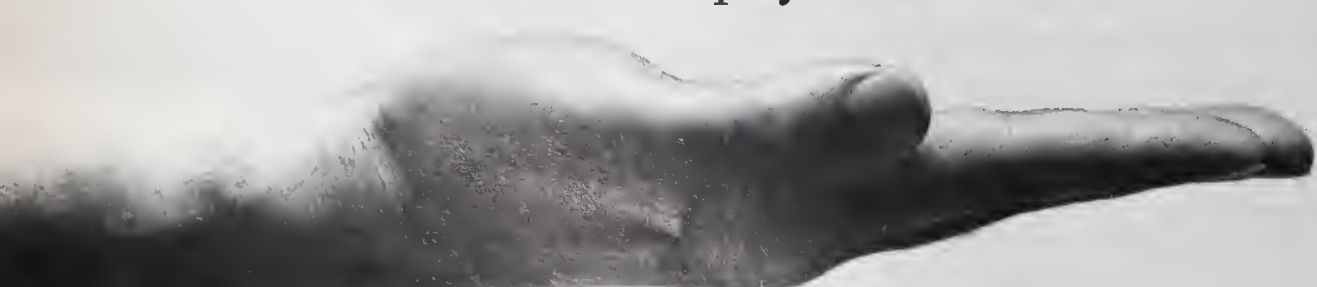
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S *President's Page* **D**



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The Endowment Association exists solely to help meet the loan needs of medical students at the University of South Dakota School of Medicine. It is administered by Staff and members of the State Medical Association.

With inflation and rising tuition costs, there are many students in need of financial support during their four years in Medical School. Surprisingly, contributions to the endowment fund have decreased the past two years, in spite of increasing need of students. As 1981 nears an end, please consider a tax-exempt contribution to your Endowment Association and keep inflation in mind as you write your check.

Address contributions (Tax Deductible) to South Dakota Medical School Endowment Association, 608 West Avenue North, Sioux Falls, South Dakota 57104.

The future doctors of South Dakota can use your help. Thank you so much.

Sincerely yours,

Bruce Lushbough, M.D., President
South Dakota State Medical Association

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The Need For "P.R. Thinking" In Your Medical Practice*

Leif C. Beck, LL.B., CPBC,
Vasilios J. Kalogredis, J.D., CPBC,
Geoffrey T. Anders, CPA, J.D. and
Dorothy R. Sweeney**

Impending Economic Crunch

Physicians are by their nature not particularly interested in the rather disdained fields of marketing, advertising and "public relations". They decided on their lines of work for reasons tied to technical interest and service to others, and these reasons are somewhat incompatible with the thought of "selling". In fact, as we have pressed some of our medical clients to more affirmatively market their practices, the responses have often been blank stares.

Well, the times are changing and it behooves physicians to change with them. Government studies and responsible articles follow one upon the other to demonstrate the increasing oversupply of physicians, expected to reach large numbers during this decade. The situation has in many specialties and areas already caused practice growth to fall off. Income increases appear these days to be limited mainly to fee changes, as the number of patients seen stays level or diminishes.

Doctors with busy, successful practices just don't seem interested in these symptoms. Some of them essentially respond that they are so busy rendering high-quality care to so many patients that they have no time to worry; and the concern is put beneath their professional dignity. It is, however, convenient to place oneself "above" such economic concern so long as one is earning a high income, though that

level of principle tends to bend as the economics changes.

We expect, however, that even presently successful practices will feel an economic "crunch" in the next five and ten years. Doctor advertising is beginning to appear and will increase dramatically. Newer practices will find ways to tell the community of favorable fee schedules, of willingness to participate in Blue Shield and accept Medicare assignments and even of expertise in special procedures. And specialists will concentrate heavy promotion of their characteristics to potential referring doctors, breaking down the reliability of presently comfortable single-source referral lives.

Governmental and Institutional Competition

Even before the predictions of doctor oversupply, there were some ominous warnings. Back in 1974, we wrote an article entitled "Facing Medicine's Gravest Problem",*** which included the following:

"Interestingly enough, very few physicians make any correlation between poor public relations and the ominous trend of the government to intervene in private medicine. When the possibility of such a relationship is suggested, most doctors express firm disbelief and attribute legislation on medical care to the persistent efforts of a few professional liberals. When such an element obviously exists and has had considerable influence in Congress, the simple truth of the matter is that most members of Congress voted for these measures because their constituents wanted them. While this may be a difficult concept to accept—and I know that many cynics will describe it as naive—I would venture to guess that a public referendum on National Health Insurance at this time would find

* Copyright by Authors, November 1980.

** Messrs. Beck, Kalogredis, Anders and Ms. Sweeney are the principal consultants of Management Consulting for Professionals, Inc., Bala Cynwyd, Pa.

***Facing Medicine's Gravest Problem", by Leif C. Beck, *Pennsylvania Medicine*, June, 1974, p. 32, which quoted extensively from an article by Thomas F. Zirkle.

overwhelming public support for the program with perhaps a large footnote condemning the professions for the many well-publicized abuses which have appeared in the press.

You may disagree with this conclusion; yet it seems inevitable to me that government intervention in medicine will directly and accurately reflect the state of discontent between doctor and patient."

The real solution to the governmental threat is almost totally in the hands of the private doctor who deals with his patients on a person-to-person basis. He must convey to each patient that he is the most competent and most thoughtful available physician for him or her—that he satisfies the patient's need better than the government-supported HMO or the large multi-specialty clinic. **It matters not in this respect whether that doctor actually is "best", for the patient's selection will be based only on perceptions.**

So "marketing" and "public relations" are the essential ingredients in protecting the presently successful doctor against these other likely encroachments on his practice. The HMO is in fact gobbling up large numbers of patients otherwise treated by solo doctors and small groups. And the clinic-sized multi-specialty groups are keeping patients who cease being referred to independent non-clinic specialists.

The HMO's employ experienced, well paid marketing experts; those people are key members of prepaid practices' executive staffs. Similarly, we expect other large medical groups to begin hiring public relations experts during this decade. The architectural, accounting and law professions are already seeing this phenomenon, and there is no reason to assume that medical groups will not follow the same path.

Unless the private practitioner needs these governmental and institutional inroads by having "satisfied customers", his practice will be chipped away. So he will face competition both from the oversupply of small practices and from the growth of the big organizations.

We urge that the privately practicing physician anticipate the problems and start now to protect his practice. It may be too late to begin thinking about "public relations" and "marketing" when his practice has already begun to sputter.

What Can The Doctor Do?

Doctors can begin meeting the described threats first by simply tuning their thought processes to the importance of "P.R.". They can still practice the highest quality medicine, but they must be critical whether their patients **appreciate** the quality and the concern—the doctors must care that their well-treated patients are in fact "satisfied customers".

As to office practice, there is so much to consider. The physician must be sure to provide a comfortable office environment, both as to the physical facility and in having helpful, hospitable assistants. Employees who are unsympathetic or unfriendly may have to be weeded out.

Physicians must also recognize their patients' concern for promptness. It just isn't fair or considerate to keep patients waiting simply because the doctor overbooked his schedule, and when he is unavoidably late he can at least give the courtesy of letting them know the compelling reasons for delay. Patients consider their time as valuable as the doctor's, so some "P.R." can help them accent a long wait more sympathetically.

Billings, collection follow-up and insurance handling can be conducted so the patient will appreciate and understand an office's routines. Especially as so many doctors elect not to participate in Blue Shield or accept Medicare assignments, they must be sure their patients have been told and understand the process.

There are a variety of other public relations steps which some doctors are taking and which we expect to grow in the next few years. Here are a few of them.

Patient Information Booklets

Many physicians have drafted informative brochures describing their practices and their basic arrangements of importance to patients. Such "patient information booklets" are timesaving devices for a doctor's office, and one estimate suggests they will reduce incoming telephone calls by 20% to 30%. Just as importantly, however, the booklet can be a courtesy to the patient, attempting to reach out and communicate with him or her before misunderstandings might arise.

One very good use of the booklet is with new patients. They typically know little about the doctor(s) or the office and its policies, in which case some sort of advance indoctrination can help prevent problems. Consider, for example, a patient's surprise at an office's disproportionately high first office visit fee, at its policy for payment "over-the-counter" or at its non-participation in Blue Shield and/or refusal to take Medicare assignments.

We therefore suggest a routine of mailing the patient information booklet to each new patient as soon as the first office visit is scheduled. The receptionist need merely ask the patient's address as she makes the appointment and then promptly send out the booklet. A small preprinted card might be attached saying something like:

"We look forward to meeting you at the appointment made for (date and time). For your information and review before your visit, the enclosed pamphlet describes our practice and some of its

policies. If you have any questions, please feel free to call us right away."

Some doctors complain that patient information booklets lose their effectiveness as patients misplace them or disregard them. We do not find that to be true enough to justify stopping distribution of the booklets. Furthermore, to the extent they are shown to patients' friends, the information pamphlets become low key advertising of one's practice.

Patient Newsletters

Physicians can take some advice from the dental profession. An increasing number of dentists now prepare and send quarterly newsletters to all their patients. These newsletters are usually written fairly casually by the doctors and their employees to report on matters of interest related to their practices. An underlying purpose of course, is to remind the patients of the doctor's interest, to hold the patient's allegiance and generally to serve as good "P.R."

Professionally, a newsletter can satisfy the growing emphasis on "prevention". A doctor can remind his patients of certain health matters for their benefit, particularly including any new information in his specialty that may be relevant. This is the age of prevention, and the newsletter may be one way a physician can help raise the level of his patients' health consciousness.

We predict that in the next five years there will be a rash of physicians' newsletters addressed to patients. Developing a well-planned, high quality publication now might help put you ahead of this likely trend in which the beneficiaries will be both your patients and your practice.

We have even heard of a physician who leaves a supply of his newsletters at the local drug and health food stores. While this may presently repel most of our readers as unprofessional, they will nevertheless find such market-plays increasingly threatening to their practices over the next few years.

Patient Questionnaires

It is commonly recognized that a physician will be the last person to learn of patient complaints about his office, its level of courtesy, the business arrangements and the like. Patients fuming over long waits in a dingy reception room, for example, tend not to express their annoyance when they are finally examined by the doctor. Similarly, displeasure at an assistant's snippiness or at the handling of one's Medicare form will only occasionally be directed personally at the doctor.

While this fact insulates the physician from any "feel" for his practice's patient relations, it is also annoying to his patients. They come to assume either that the doctor doesn't structure his practice to serve them thoughtfully or that he doesn't care, or both.

Some offices, therefore, periodically prepare a brief

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*Data on file Parke-Davis Marketing Research Dept.
**Based on total prescriptions filled for hemorrhoidal
preparations during the first three quarters of 1980.
The National Prescription Audit, IMS America Ltd.,
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PD-400-JA-0146-P-1 (1-81)

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Vaginal Uses—Comforting as an adjunct in postoperative care after episiotomies and other vaginal surgery or when relief from vaginal itching, burning or irritation is required.

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ANUSOL-HC® CREAM

Rectal Cream with Hydrocortisone Acetate

Caution: Federal law prohibits dispensing without prescription.

Description: Each Anusol-HC Suppository contains hydrocortisone acetate, 10.0 mg; bismuth subgallate, 2.25%; bismuth resorcin compound, 1.75%; benzyl benzoate, 1.2%; Peruvian balsam, 1.8%; zinc oxide, 11.0%; also contains the following inactive ingredients: dibasic calcium phosphate, and certified coloring in a hydrogenated vegetable oil base.

Each gram of Anusol-HC Cream contains hydrocortisone acetate, 5.0 mg; bismuth subgallate, 22.5 mg; bismuth resorcin compound, 17.5 mg; benzyl benzoate, 12.0 mg; Peruvian balsam, 18.0 mg; zinc oxide, 110.0 mg; also contains the following inactive ingredients: propylene glycol, propylparaben, methylparaben, polysorbate 60 and sorbitan monostearate in a water-miscible base of mineral oil, glyceryl stearate and water.

Anusol-HC Suppositories and Anusol-HC Cream help to relieve pain, itching and discomfort arising from irritated anorectal tissues. These preparations have a soothing, lubricant action on mucous membranes, and the antiinflammatory action of hydrocortisone acetate in Anusol-HC helps to reduce hyperemia and swelling.

The hydrocortisone acetate in Anusol-HC is primarily effective because of its antiinflammatory, antipruritic and vasoconstrictive actions.

Indications and Usage: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain, itching and discomfort in: external and internal hemorrhoids, proctitis, papillitis, cryptitis, anal fissures, incomplete fistulas, pruritus ani and relief of local pain and discomfort following anorectal surgery.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

Contraindications: Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

Warnings: The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

Precautions: General: Symptomatic relief should not delay definitive diagnoses or treatment.

Prolonged or excessive use of corticosteroids might produce systemic effects.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Anusol-HC is not for ophthalmic use.

Pregnancy

See "WARNINGS"

Pediatric Use

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

Dosage and Administration: Anusol-HC Suppositories—

Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain comfort with regular Anusol Suppositories.

Anusol-HC Cream—Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

How Supplied: Anusol-HC Suppositories—boxes of 12 (N 0071-1089-07) and boxes of 24 (N 0071-1089-13) in silver foil strips with Anusol-HC printed in black.

Anusol-HC Cream—one-ounce tube (N 0071-3090-13) with plastic applicator.

Store between 59°-86°F (15°-30°C).
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questionnaire asking the patients to evaluate how they were handled. The form might be available at the reception desk for each patient to take with him or her upon leaving the office, or it could be mailed out with each bill at month-end. It would ask a variety of questions about the waiting room, the various assistants' courtesy, the length of waiting time, the doctor's responsiveness in answering questions and the like.

Such a questionnaire should be drafted by the doctor(s) and the office manager as best suits that specific practice's concerns although assistance of an independent consultant/advisor can be helpful. The benefits of such a questionnaire, of course, will be several. First, it will help the doctor and his staff learn how they can improve their own level of responsiveness to patients; it will help them structure the practice more competitively.

Secondly, a questionnaire tells a physician's patients that he really does care how they are handled in their encounters with him, his physical office and his staff. Just the fact of asking (assuming he gives attention to the answers) will thus be a positive item in a doctor's effort to market his practice.

Drug Information Pamphlet

A good client of ours recently developed a special patient information pamphlet entitled "Drug Effects and Side-Effects". It is a simple preprinted folder, containing the title and the doctor's name, address and phone number on the cover. On one inside page is a brief description, in the doctor's own personalized words, telling why the booklet is being provided. And on the other inside page will be stapled the specific drug's description.

This doctor has drafted his own separate discussion of each drug he commonly prescribes. Each description is separately preprinted so the page for whichever drug he prescribed can be stapled into the folder and handed to the patient.

The patient response has been favorable. From a medical standpoint, the pamphlet has helped this physician assure that his patients are well informed as to their prescribed drugs—a form of risk control. And from a "P.R." standpoint, his patients have another indication that he really cares about their treatment and their information. He thus comes off better on this point than do the other doctors in his specialty who do not give this "something extra".

Practice Development Advisors

We were surprised to learn recently that a company has been started in Washington, D.C. to specifically counsel medical practices on building their patient loads. They would study the doctor and his office to help him market his services more ef-

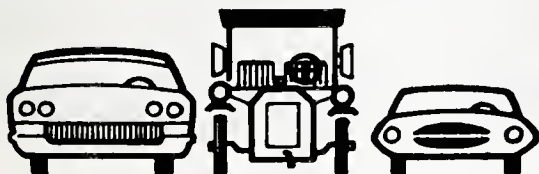
fectively, presumably including emphasis on the doctor's own personality, communication skills and the like. While we are not presently acquainted with this new company or its principals (a physician and a management consultant), we see its presence as one more example of the times.

Conclusion

Private medical practice as most of our clients know it is at a crisis point. Between the continuing supply of more potentially competing physicians and the expansion of HMO's and large clinics, the pressures on successful practitioners will grow during this decade. Practices that are now oblivious to these pressures may well find their patient bases become threatened and perhaps even begin to shrink.

These pressures will accelerate medical practices' interest in and use of marketing concepts—of "P.R.". Doctors who presently disdain such thought as being below their professional quality may well find themselves forced by economics to change their thinking, or else their practices will be chipped away by others who are more open-minded.

The time to deal with change is before its too late. We believe our readers and clients should stay ahead of the trend, developing real public relations/marketing thought now. We hope the described examples of such emphasis will be helpful, and we expect to see many more examples over the next few years.



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S **D** *Council Meeting Highlights*

1. **NATIONAL HEALTH SERVICE CORP.** The Council established the following procedure for commenting on applications for the placement of NHSC personnel in South Dakota. The information will be submitted to the Councilor(s) of the district society involved along with a request that he/they formulate comments and submit them to the executive office within ten days. Then the state office will forward those comments to the NHSC within the thirty day limit for responses.
2. **RETIRED LIVE'S RESERVE.** The Council accepted the recommendation of the Commission on Internal Affairs, Communications and Liaison to approve the Retired Live's Reserve program for those South Dakota physicians eligible to participate. This program allows medical corporations to provide additional benefits to its physician members upon retirement.
3. **USD SCHOOL OF MEDICINE.** Following an update on the school by Dean Hollerman, the Council took action to recommend that Council members, if they are acquainted personally with any of the Board of Regents on an individual basis, reiterate the SDSMA's position of support for the USDSM and give them accurate updated information on the school.
4. **COMMUNITY HEALTH CENTERS IN SOUTH DAKOTA.** The Department of Health and Human Services submitted a list of current community health centers in South Dakota and requested comments from the SDSMA on any or all of these projects. The Council recommended continued funding for the following Community Health Centers: 1) Miner-Hamlin, Howard, SD 2) Tri-county Health Care, Westington Springs, SD 3) Indian Health Management, Rosebud, SD 4) Northwest South Dakota Rural Health Service, Faith, SD 5) Eagle Butte Community Clinic, Eagle Butte, SD.
5. **EAGLE BUTTE PHS VS NANCY O'CONNOR, M.D.** Dr. O'Connor submitted material outlining her concerns for the medical care provided at the Eagle Butte PHS and the problems which she has encountered. The Council took action to refer this material to the USPHS, the Surgeon General of the United States, the President and South Dakota's congressional delegation and request an investigation into this matter. ■

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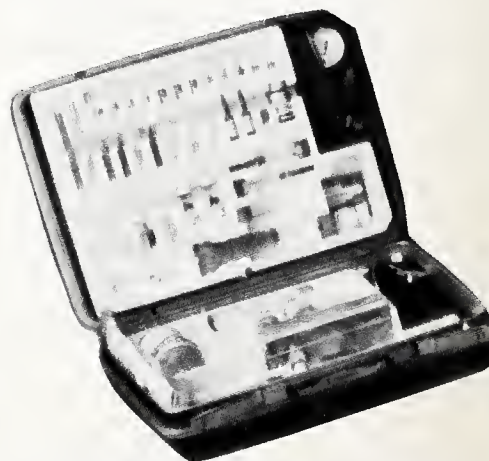
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